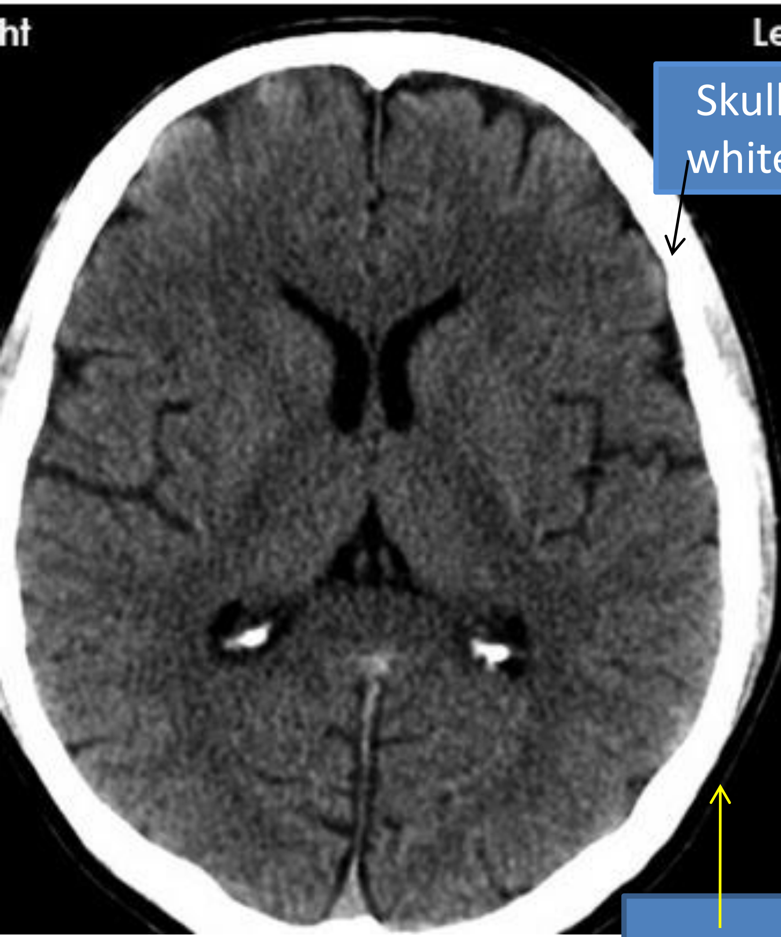
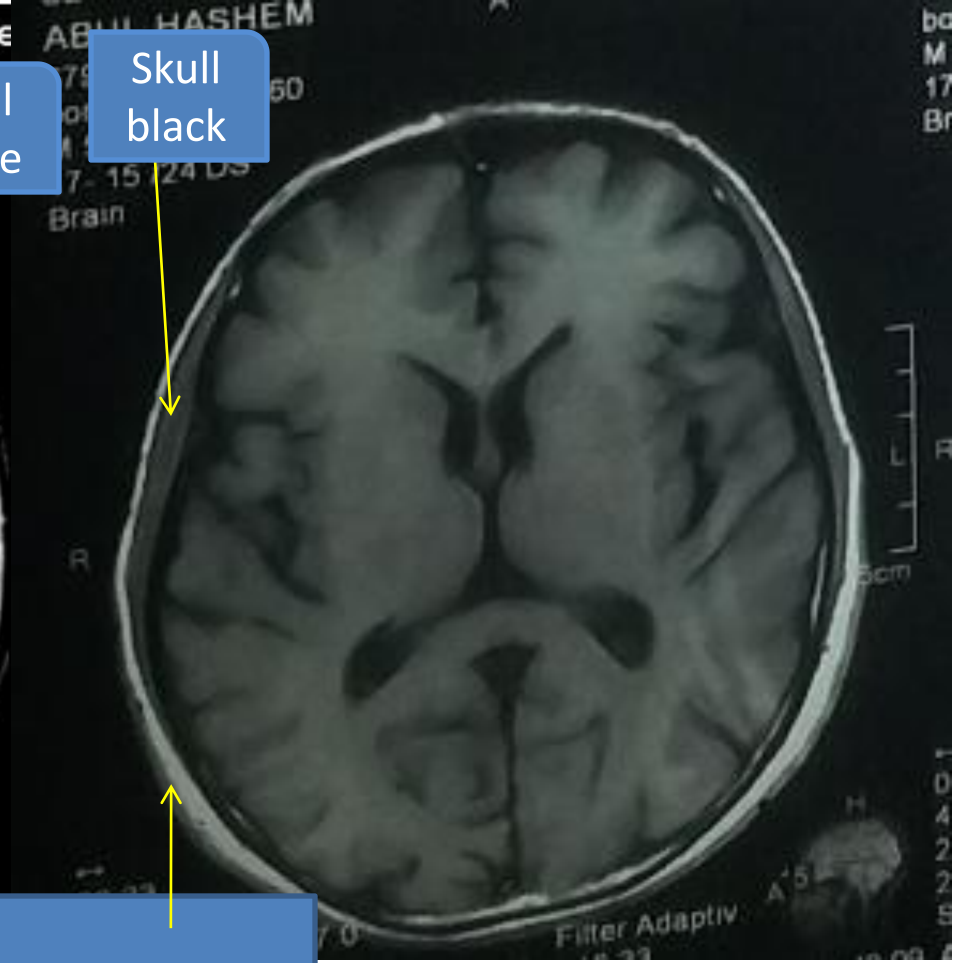
MRI OF BRAIN

DR . SHAHIDULLAH SHAMOL
FCPS (MEDICINE)



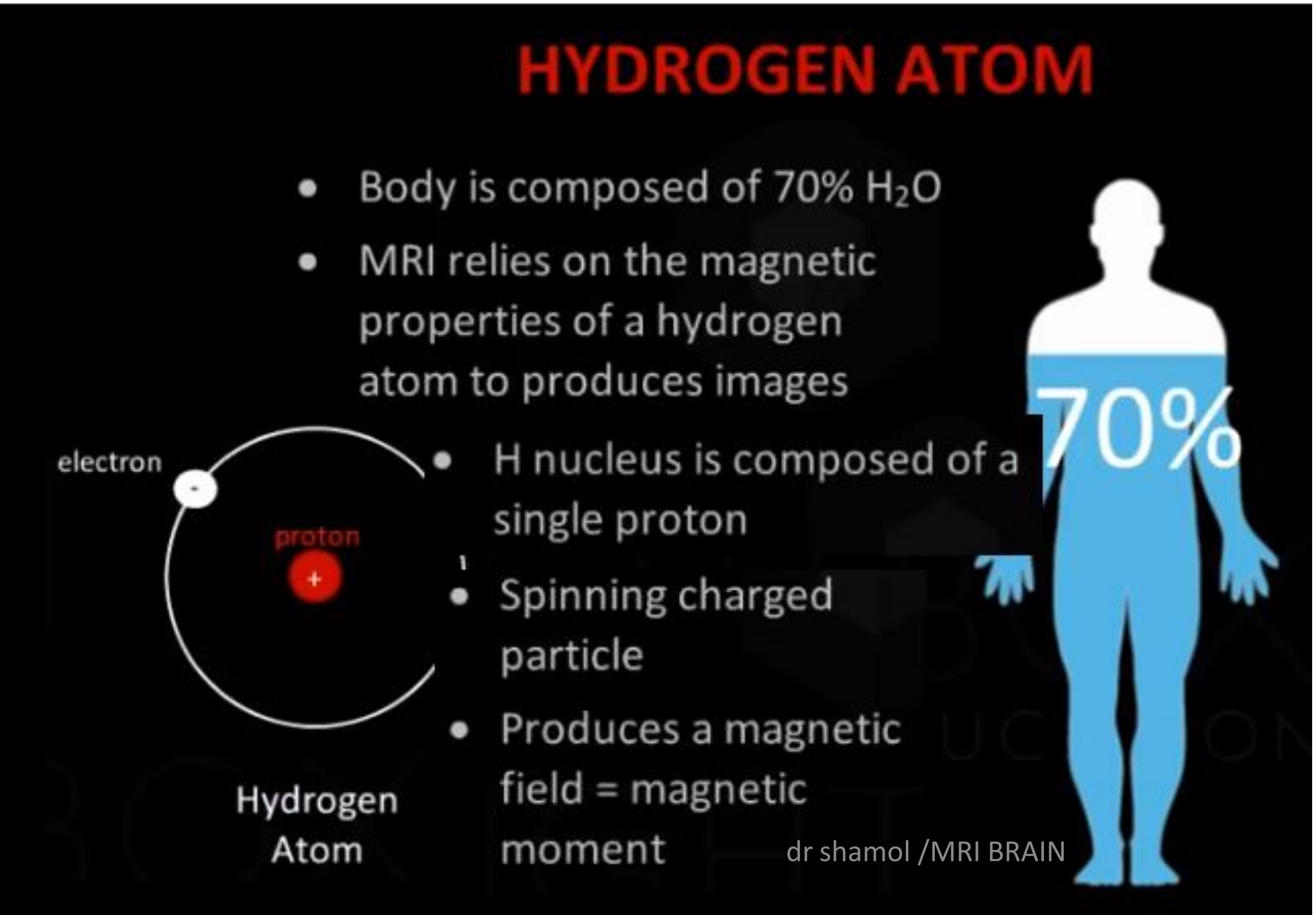


Subcutaneous fat

CT
In CT you will white rounded ring of skull bone

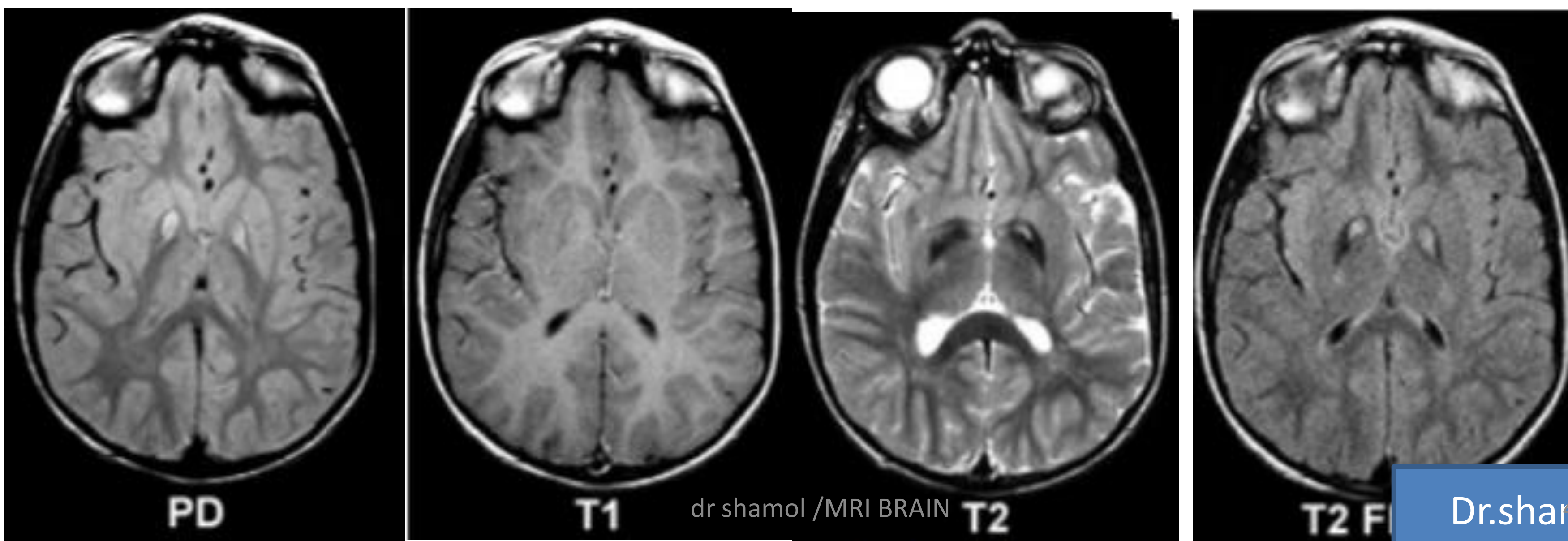
MRI T1
In MRI-T 1 white rounded ring of skull bone will not visible
The thin rounded white ring you see it is subcutaneous fat

- ❖Hydrogen is the simplest element with atomic number of 1 and atomic weight of 1
- ❖When in ionic state (H+), it is nothing but a proton.
- ❖Proton is not only positively charged, but also has magnetic spin (wobble)
- ❖We are magnets but we can’t act like magnets?
- ❖Because The protons (i.e. Hydrogen ions) in body are spinning in a hapazard fashion, and cancel all the magnetism. That is our natural state
- ❖MRI utilizes this magnetic spin property of protons of hydrogen to elicit images

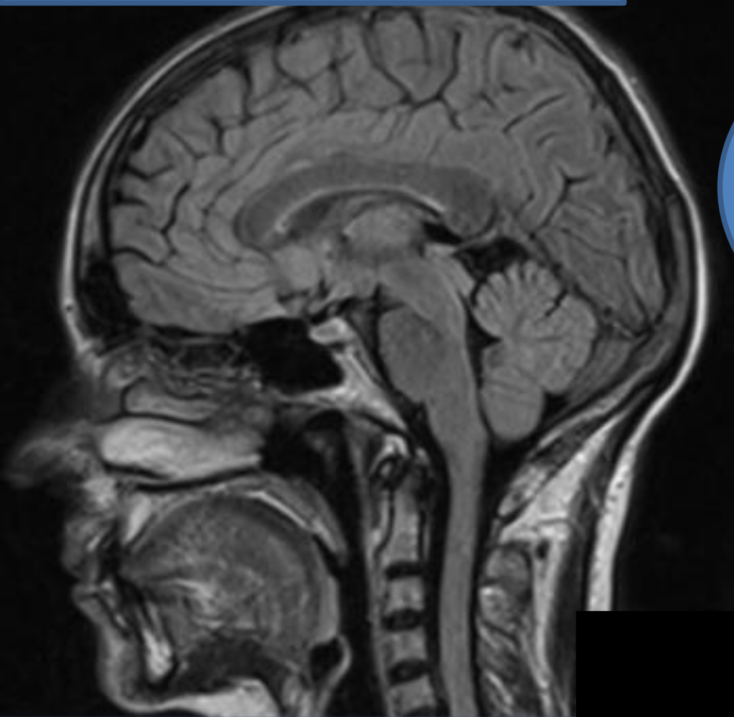


MRI creates images by picking up different signal intensities from different tissues depending on the hydrogen, or more specifically, proton content of that tissue.
PD weighted: On normal MRIs (proton density or PD weighted), the greater the hydrogen content, the brighter the image.
But radiologists and radiographers can programme the MRI machine to only pick up a certain direction of proton movement.
T1: T1 is when they programme the machine to only look at the longitudinal movement of protons. T1 images are usually used to look at normal anatomical details.

T2 : T2 is when they programme the machine to only look at the transverse movement of protons and is usually used to look at pathology because most tissues involved in disease tend to have a higher water content than normal.

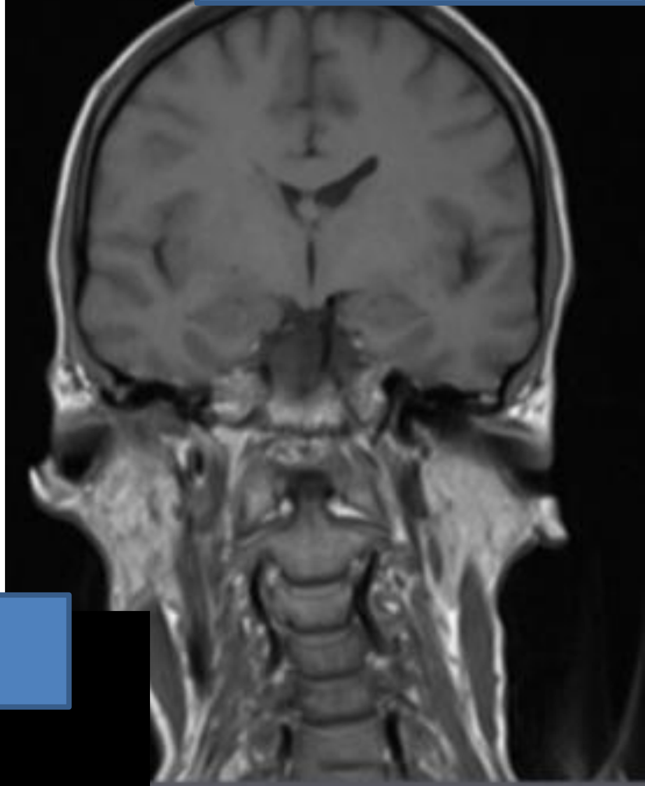


SAGIATAL



In MRI
Image is taken
in three
Plane

CORONAL



AXIAL

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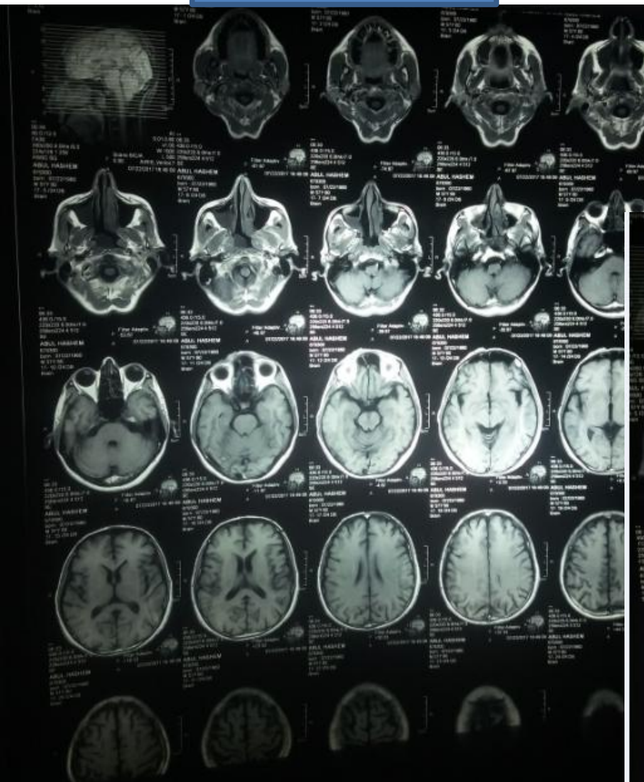
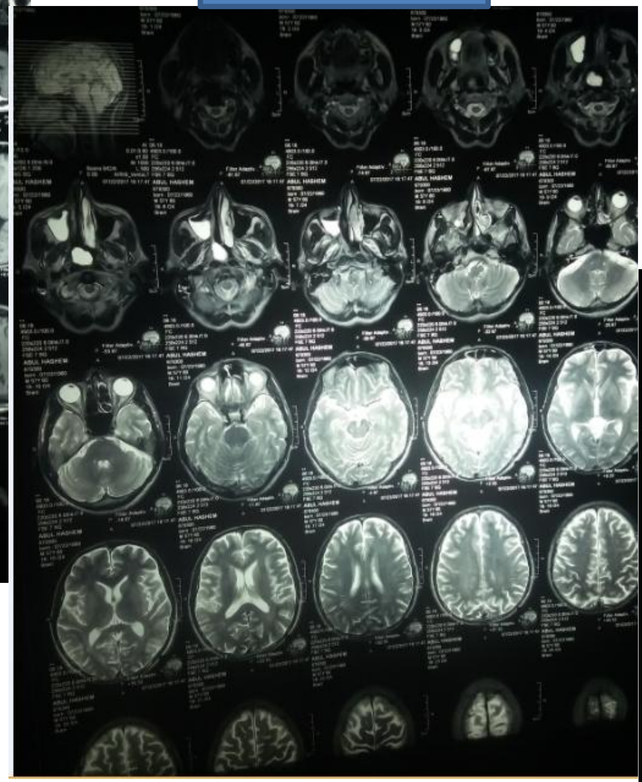
AXIAL

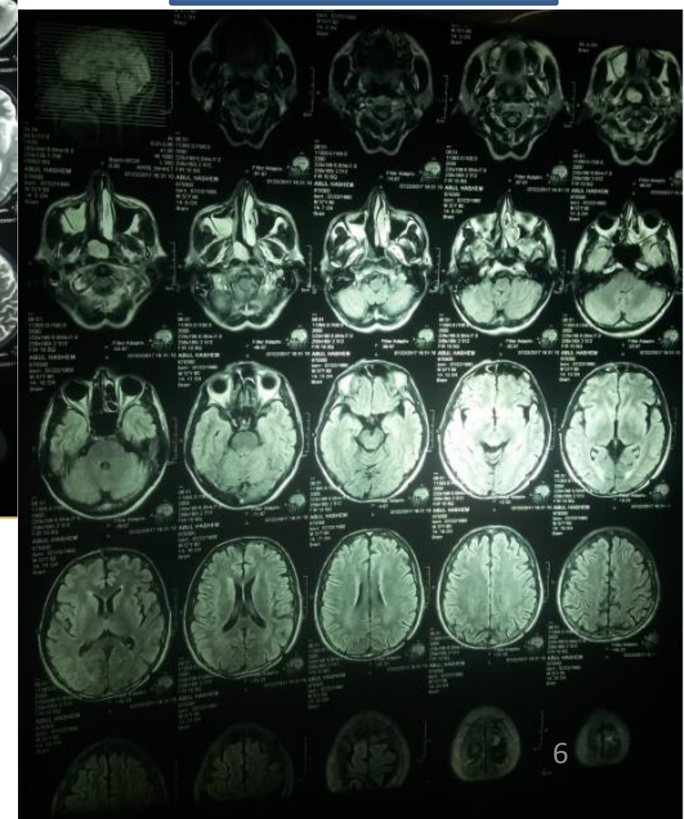
T1



T2



FLAIR



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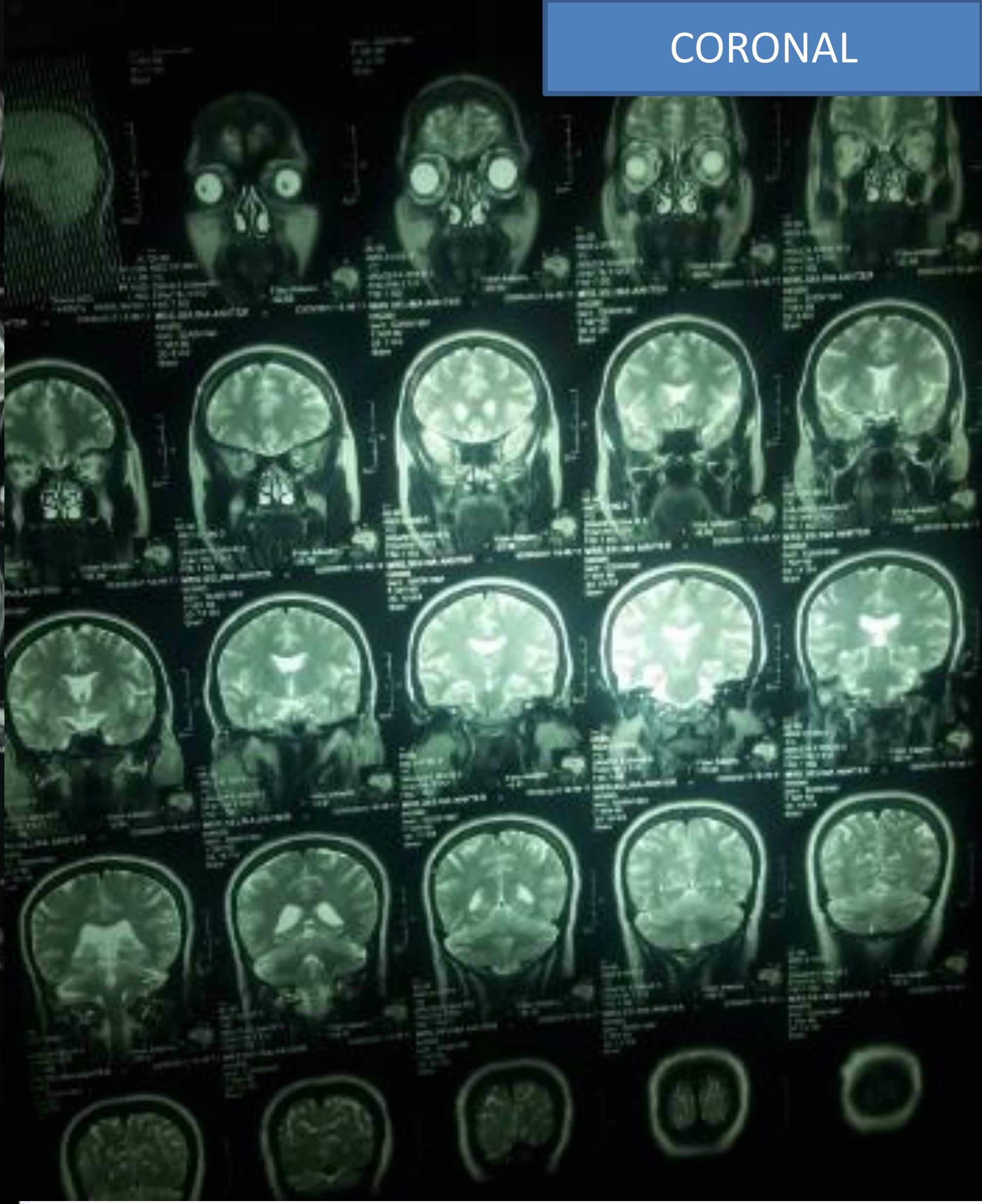
SAGIATAL

CORONAL



Intensity

When describing most MRI sequences we refer to the shade of grey of tissues or fluid with the word *intensity*, leading to the following absolute terms:

signal intensity	Color
high	white
intermediate	grey
low	black

hyperintense	brighter than the thing we are comparing it to	White
isointense	same brightness as the thing we are comparing it to	Gray
hypointense	darker than the thing we are comparing it to	Black

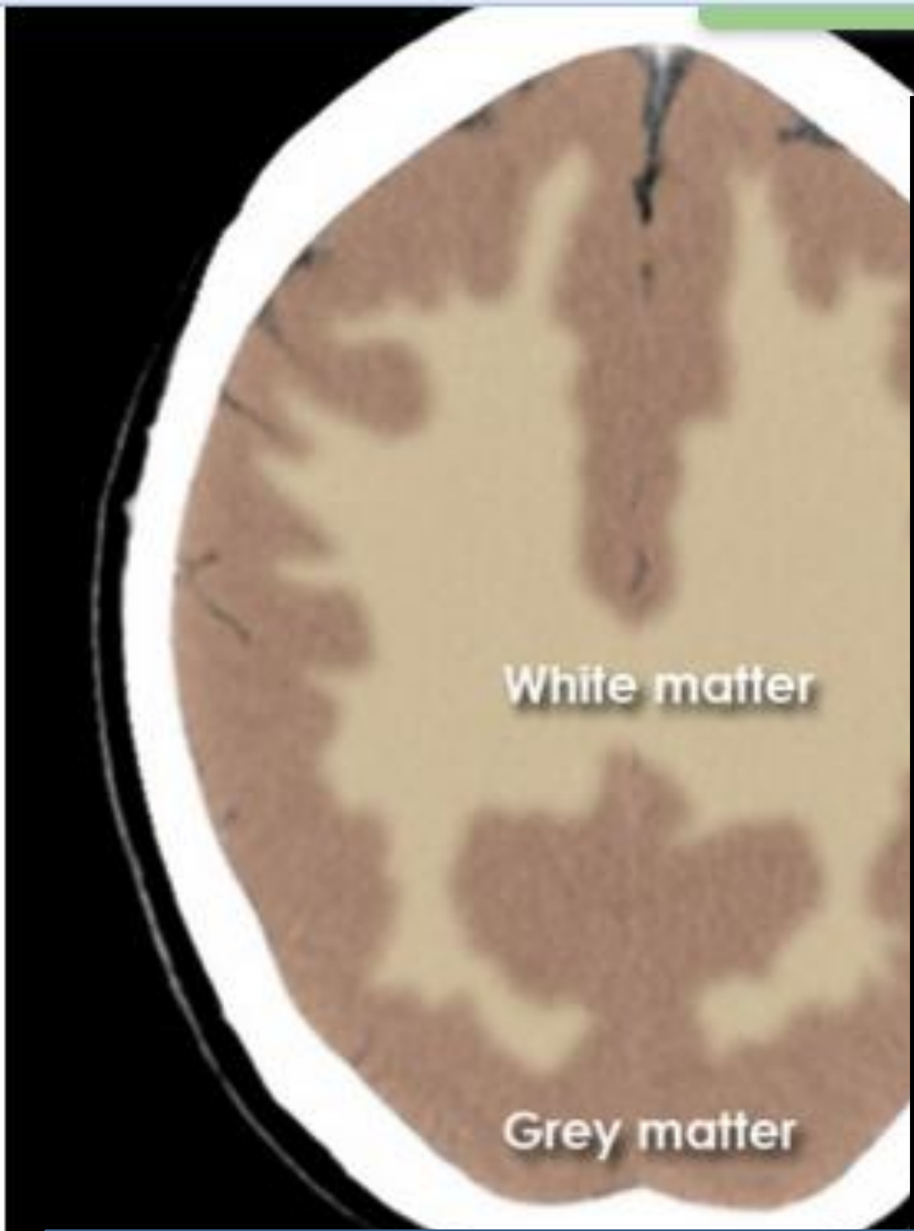
What are sequence of MRI Film :

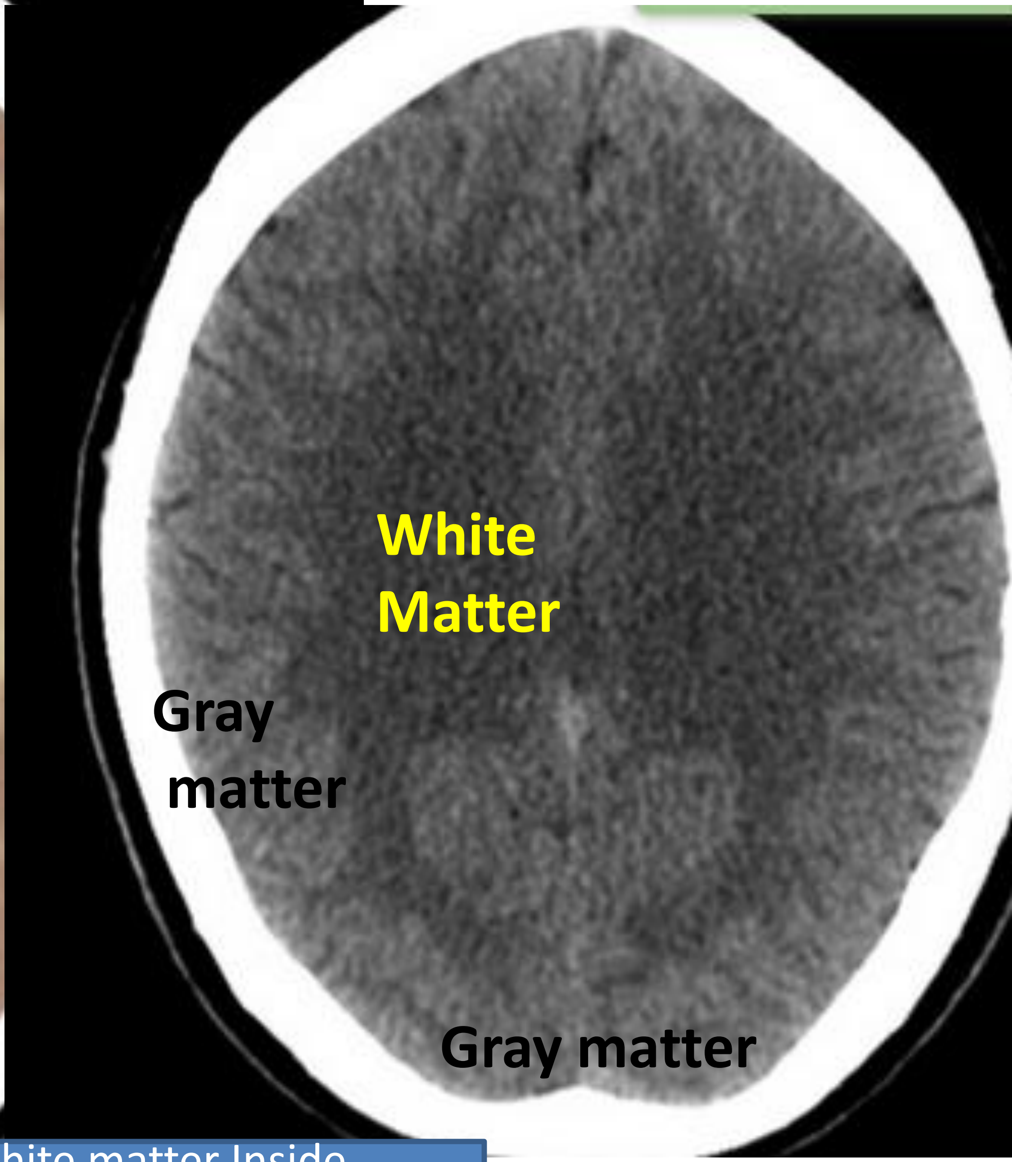
T1 weighted sequences
T2 weighted sequences
inversion recovery sequences

- ❖**fluid attenuation inversion recovery (FLAIR)**
- ❖**short tau inversion recovery (STIR)**

PD weighted sequences
Diffusion weighted sequences

- ❖**DWI**
- ❖**ADC (**Apparent diffusion coefficient maps **)**





In brain gray matter is outside and white matter Inside
In CT-white matter in black /dark and gray are less dark
The color of gray and white matter different in T1 & T2 of MRI

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T1 weighted sequences

T1 weighted sequences are part of almost all MRI protocols and are best thought of as the most 'anatomical' of images, resulting in images that most closely approximate the appearances of tissues macroscopically, although even this is a gross simplification.

The dominant signal intensities of different tissues are:	
fluid (e.g. urine, CSF):	low signal intensity (black)
muscle:	intermediate signal intensity (grey)
fat:	high signal intensity (white)
❖brain	grey matter: intermediate signal intensity (grey)
	white matter: hyperintense compared to grey matter (white-ish)

Contrast enhanced

- The most commonly used contrast agents in MRI are gadolinium based
- these agents have the effect or causing T1 signal to be increased.
- The contrast is injected intravenously (typically 5-15 mL) and scans are obtained a few minutes after administration.
- Pathological tissues (tumours, areas of inflammation / infection) will demonstrate accumulation of contrast (mostly due to leaky blood vessels) and therefore appear as brighter than surrounding tissue.

T 1

Recognition

Subcutaneus Fat is bright

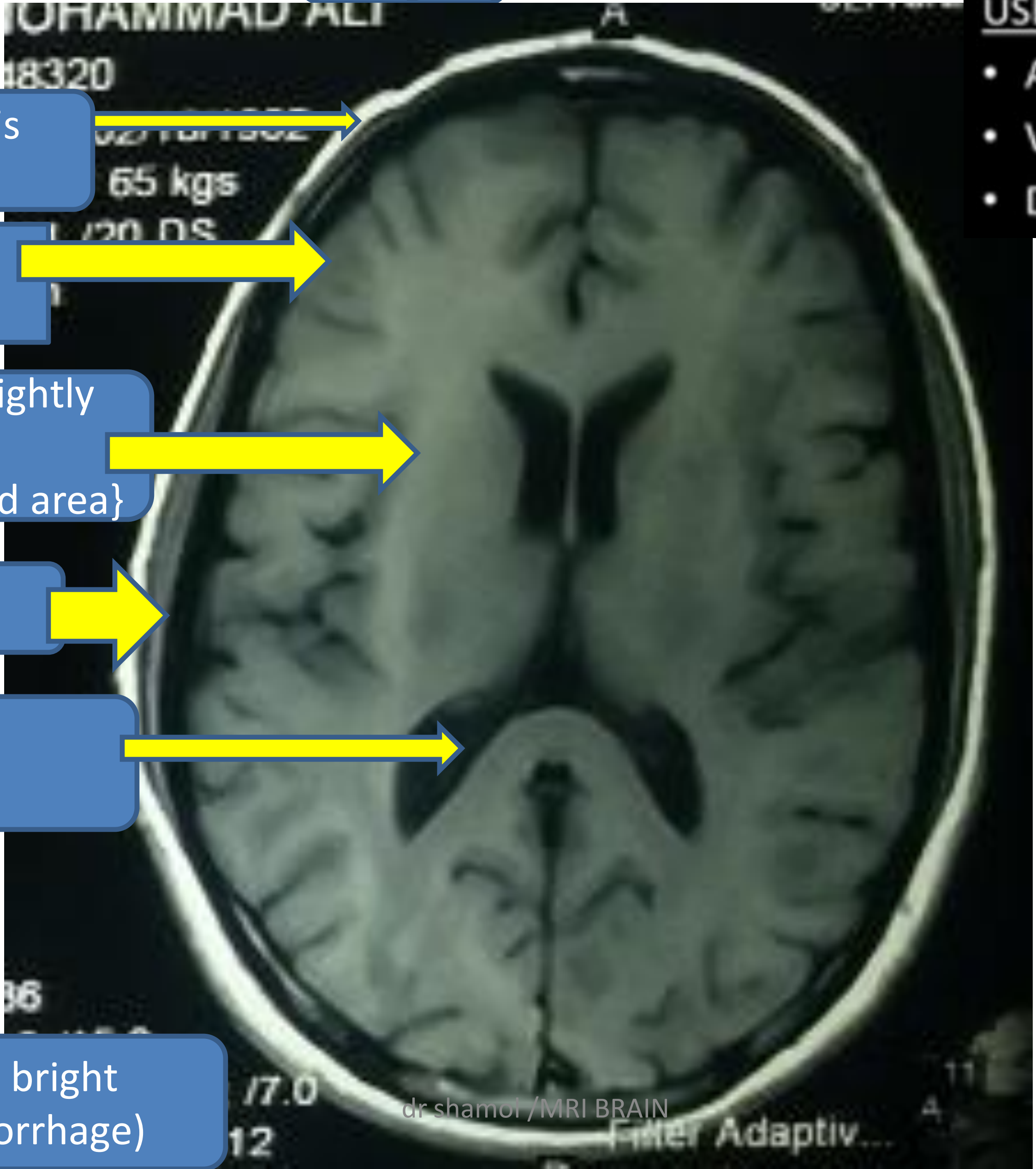
Gray mater (slightly dark)

White matter slightly bright (highly myelinated area}

Bone is dark

Water dark

New blood is bright (blood in hemorrhage)



USEFUL FOR

- Anatomic detail
- Vascular changes +C
- Disruption of BBB +C

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T2 weighted sequences

T2 weighted sequences are part of almost all MRI protocols. Without modification the dominant signal intensities of different tissues are:

The dominant signal intensities of different tissues are:	
fluid (e.g. urine, CSF):	high signal intensity (white)
muscle:	intermediate signal intensity (grey)
fat:	high signal intensity (white)
❖brain	grey matter: intermediate signal intensity (grey)
	white matter: hypointense compared to grey matter (dark-ish)

Recognition

T 2

USEFUL FOR

- Anatomic detail (CSF Spaces)
- Most lesions
- Cannot distinguish lesions from CSF

Subcutaneus Fat is bright

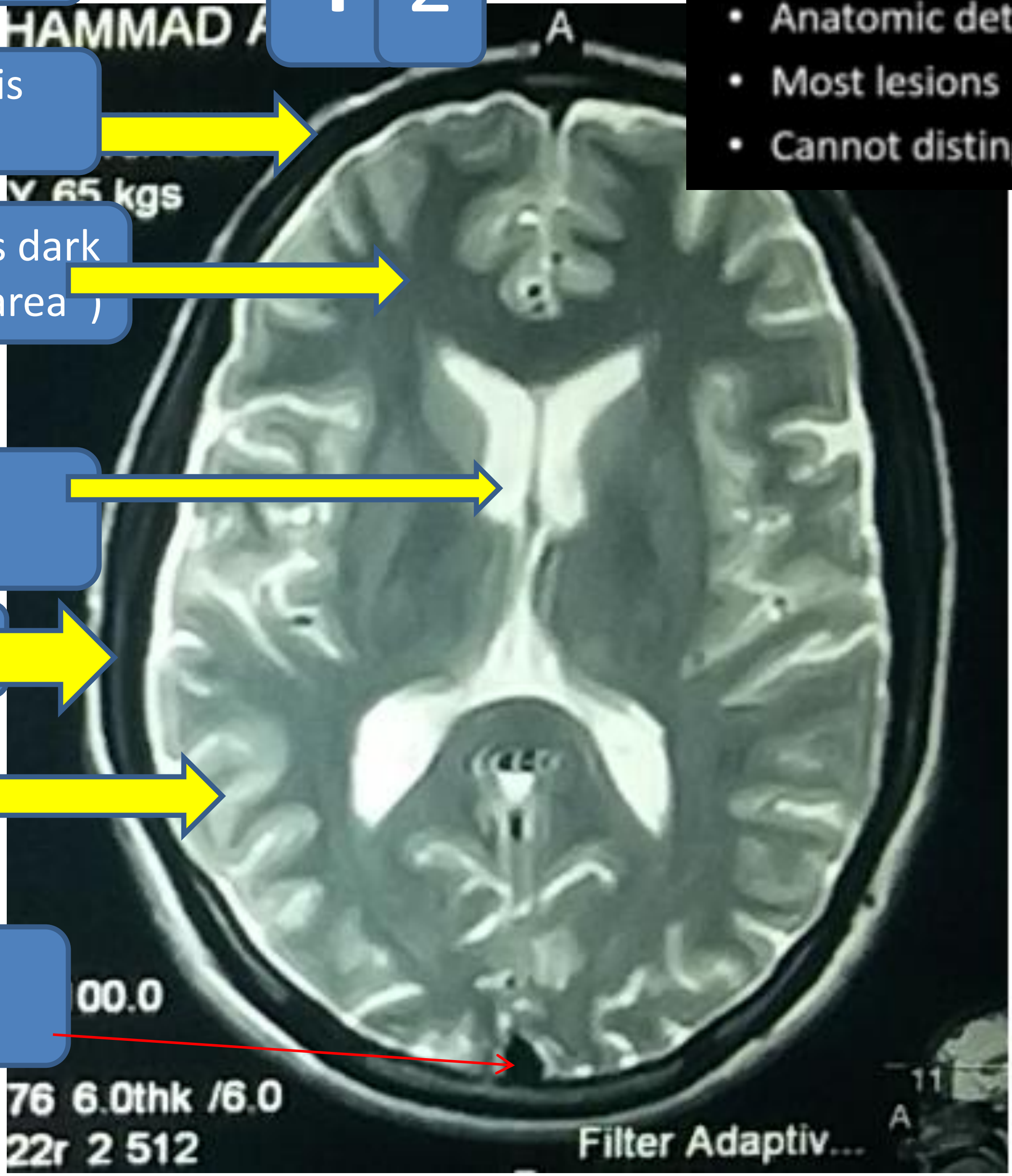
White matter /Fat is dark (highly myelinated area)

Water bright

Bone is dark

Gray mater (slightly dark)

Flow is dark (blood in vessel)



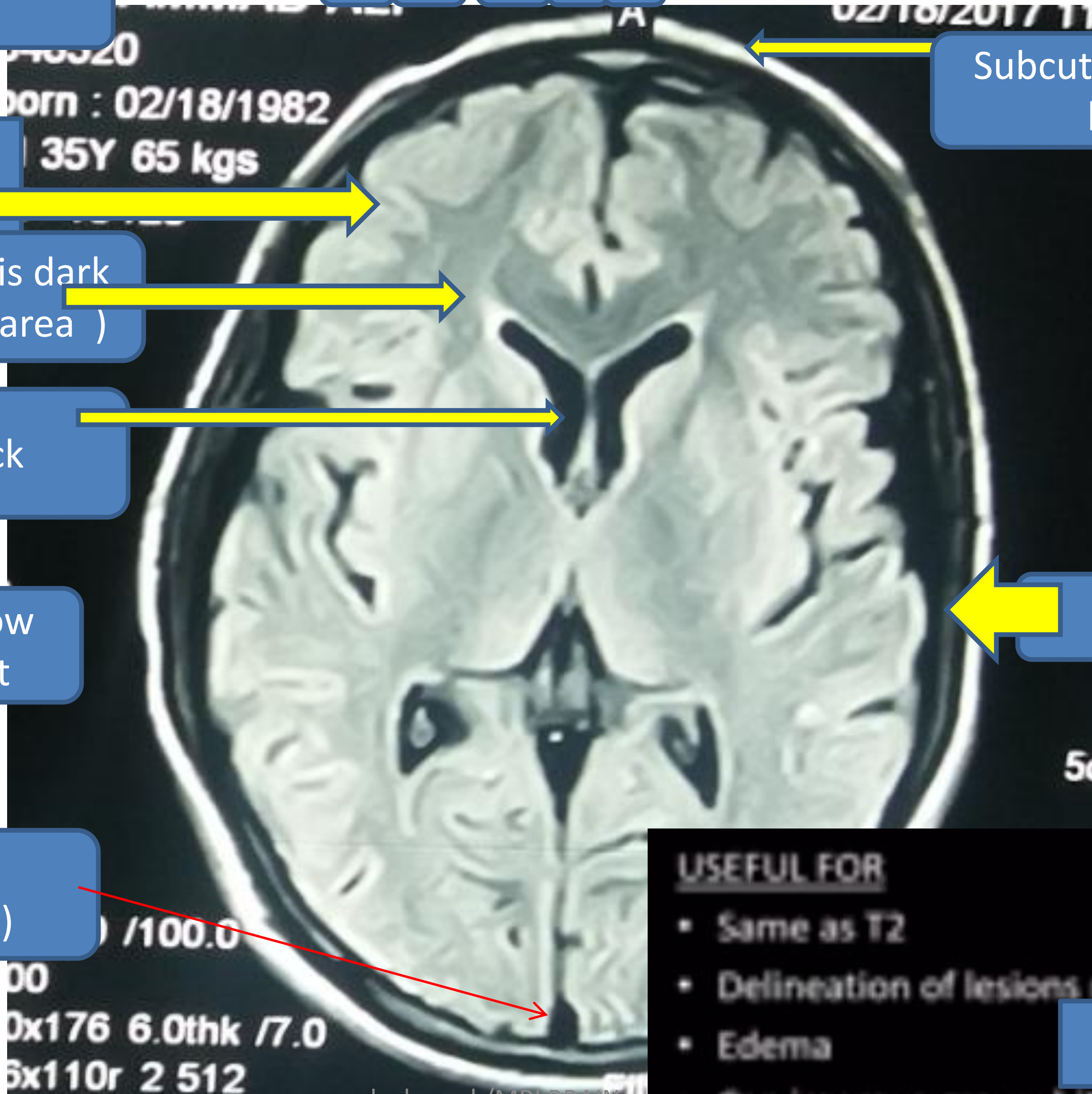
Fluid attenuated inversion recovery / FLAIR

- ❖ in the brain, we often want to detect parenchymal oedema without the glaring high signal from CSF.
- ❖To do this we suppress CSF. This sequence is called FLAIR.
- ❖Importantly, at first glance FLAIR images appear similar to T1 (CSF is dark).
- ❖The best way to tell the two apart is to look at the grey-white matter.
- ❖T1 sequences will have grey matter being darker than white matter.
- ❖ T2 weighted sequences, whether fluid attenuated or not, will have white matter being darker than grey matter.

The dominant signal intensities of different tissues are:	
fluid (e.g. urine, CSF):	low signal intensity (black)
muscle:	intermediate signal intensity (grey)
fat:	high signal intensity (white)
❖brain	grey matter: intermediate signal intensity (dark-ish)
	white matter: hypointense compared to grey matter (whitish)

FLAIR

Recognition



Subcutaneus Fat is bright

Gray mater (slightly white)

White matter / Fat is dark (highly myelinated area)

CSF/Water black

Bone is dark

Edema / non flow water is bright

Flow is dark (blood in vessel)

USEFUL FOR

- Same as T2
- Delineation of lesions near ventricles
- Edema
- Can improve grey-white differentiatio

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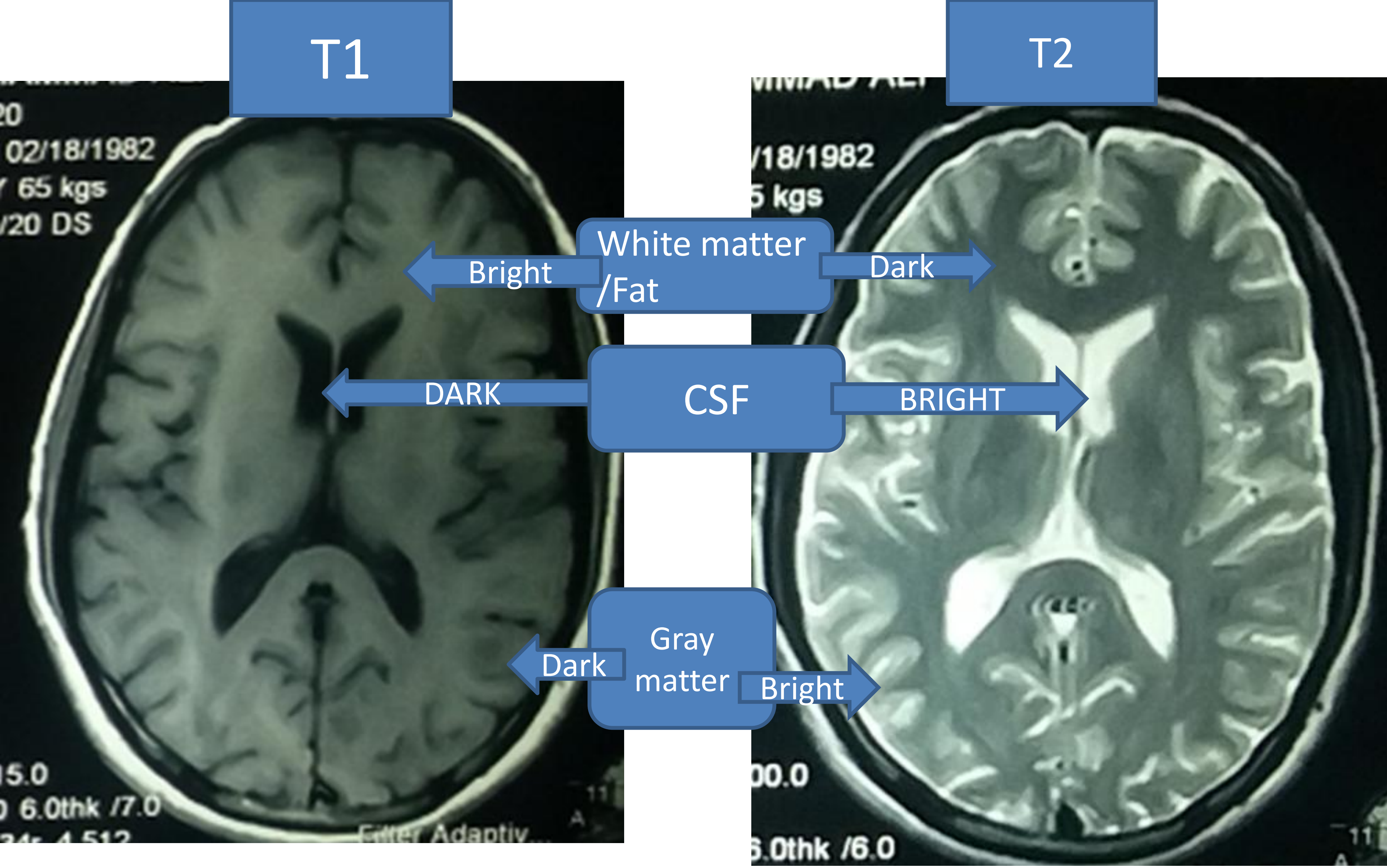
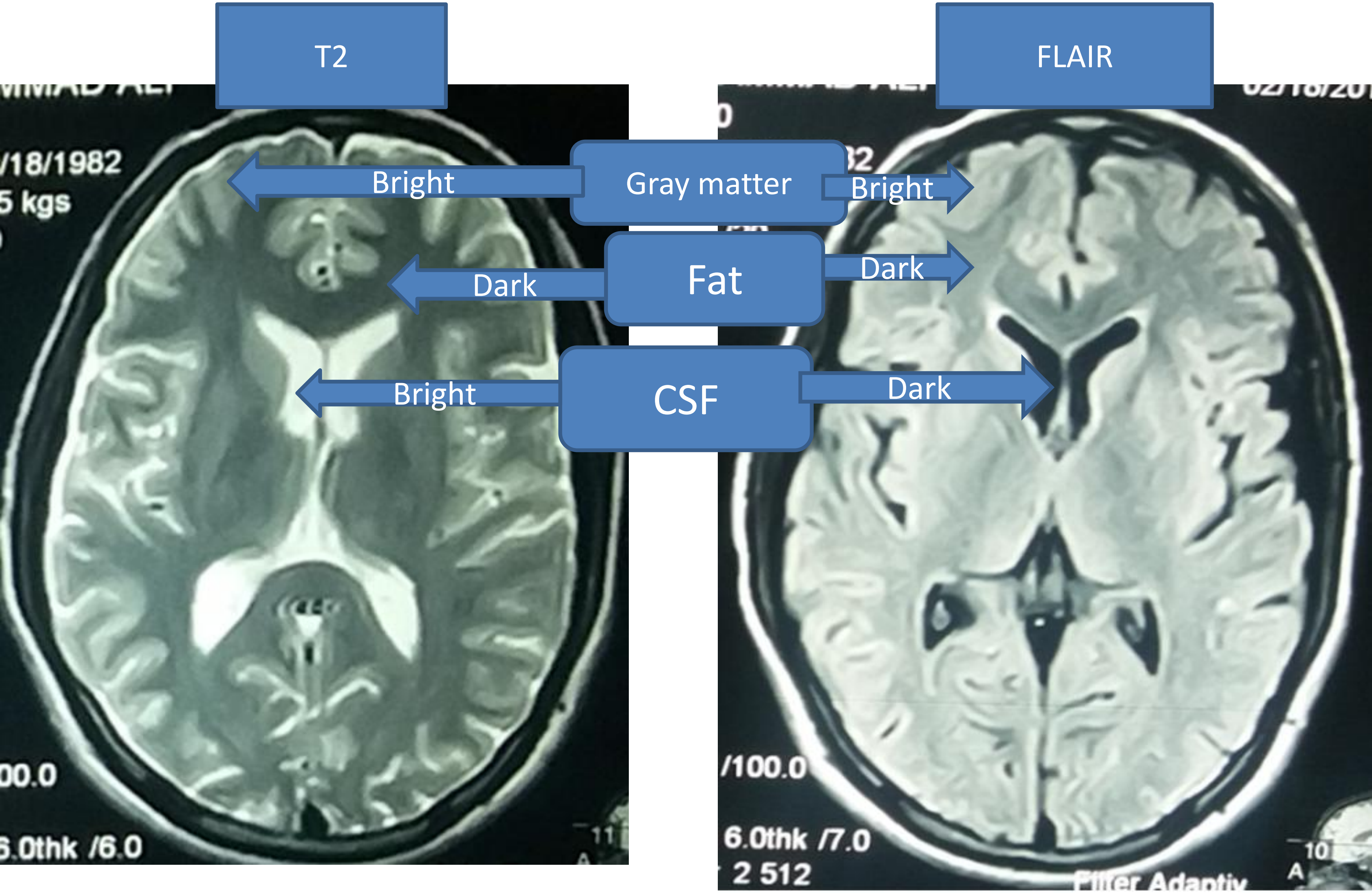
Basic changes in T1,T2 & flair


T1
T2
02/18/1982
65 kgs
/20 DS
15.0
6.0thk /7.0
Filter Adaptiv...
/18/1982
5 kgs
00.0
6.0thk /6.0
White matter /Fat
Bright
Dark
CSF
DARK
BRIGHT
Gray matter
Dark
Bright


T2
FLAIR
Gray matter
Bright
Bright
Fat
Dark
Dark
CSF
Bright
Dark
/18/1982
5 kgs
00.0
6.0thk /6.0
/100.0
6.0thk /7.0
2 512

PD weighted sequences

❖It reflect the actual density of protons; an intermediate sequence sharing some features of both T1 and T2.

❖Proton density images were extensively used for brain imaging, however they have largely been replaced by FLAIR.

❖ PD however continues to offer excellent signal distinction between fluid, hyaline cartilage and fibrocartilage makes this sequence ideal in the assessment of joints.

The dominant signal intensities of different tissues are:	
fluid (e.g. joint fluid, CSF):	high signal intensity (white)
muscle:	intermediate signal intensity (grey)
fat:	high signal intensity (white)
hyaline cartilage:	intermediate signal intensity (grey)
fibrocartilage:	low signal intensity (black)

Diffusion weighted sequences

Diffusion weighted imaging assess the ease with which water molecules move around within a tissue (mostly representing fluid within the extracellular space) and give insights into

- ❑ cellularity (e.g. tumours),
- ❑cell swelling (e.g. ischaemia) and
- ❑oedema.

The dominant signal intensities of different tissues are:

- ❖fluid (e.g. urine, CSF): no restriction to diffusion
- ❖soft tissues (muscle, solid organs, brain): intermediate diffusion
- ❖fat: little signal due to paucity of water

Typically you will find three sets of images when diffusion weighted imaging is performed:

- ✓DWI (Diffusion weighted image)
- ✓ ADC (Apparent diffusion coefficient maps) and
- ✓ B=0 images.

DWI is designed to detect the random movements of water protons. Water molecules diffuse relatively freely in the extracellular space; their movement is significantly restricted in the intracellular space. Spontaneous movements, referred to as diffusion, rapidly become restricted in ischemic brain tissue. During ischemia, the sodium - potassium pump shuts down and sodium accumulates intracellularly. Water then shifts from the extracellular to the intracellular space due to the osmotic gradient. As water movement becomes restricted intracellularly, this results in an extremely bright signal on DWI. Thus, DWI is an extremely sensitive method for detecting acute stroke.

DWI“

Is an isotropic T2 weighted map as it represents the combination of actual diffusion values and T2 signal.

It is a relatively low resolution image with the following appearance:

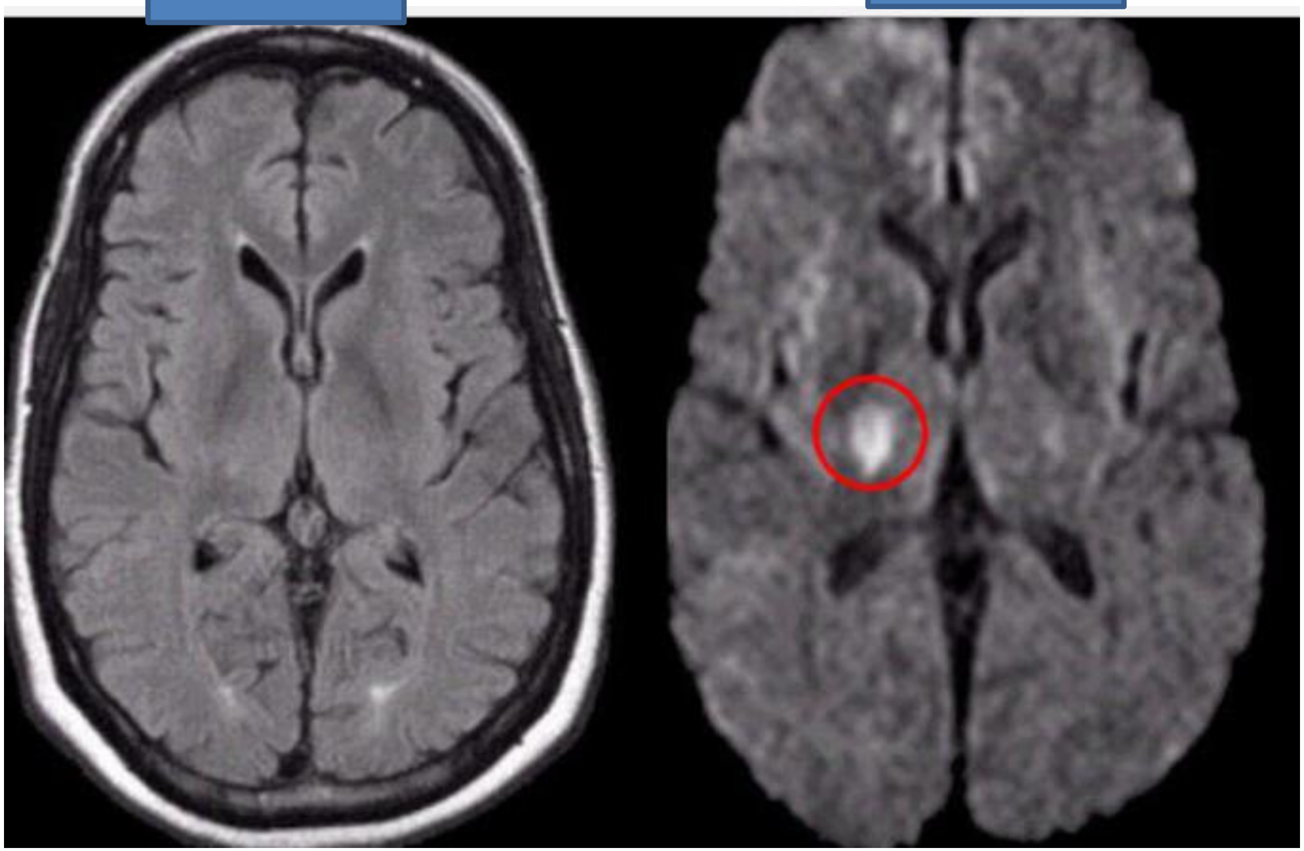
grey matter:	intermediate signal intensity (grey)
white matter:	slightly hypointense compared to grey matter
CSF:	low signal (black)
fat:	little signal due to paucity of water
other soft tissues	intermediate signal intensity (grey)

Acute pathology (ischaemic stroke, cellular tumour, pus)	usually appears as increased signal (white)denoting restricted diffusion.

However (and importantly) because there is a component of the image derived from T2 signal some tissues that are bright on T2 will appear bright on DWI images without there being an abnormal restricted diffusion. This phenomenon is known as T2 shine through.

FLAIR

DWI



ADC

Apparent diffusion coefficient maps (ADC) are images representing the actual diffusion values of the tissue without T2 effects.

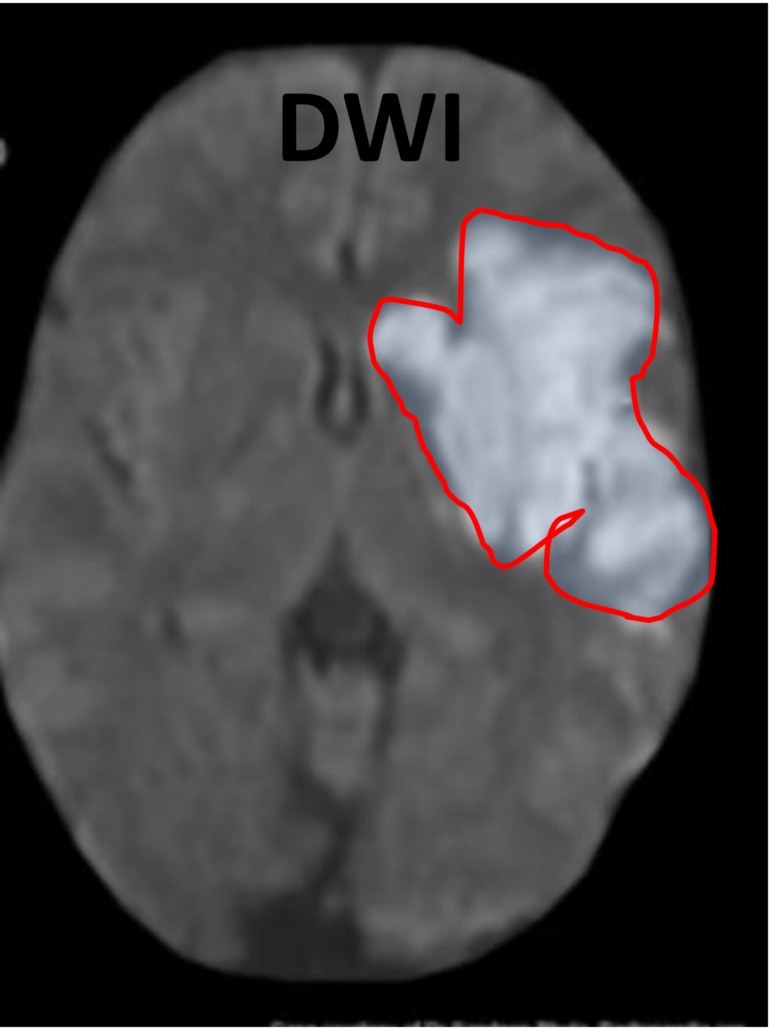
They are therefore much more useful, and objective measures of diffusion values can be obtained, however they are much less pretty to look at.

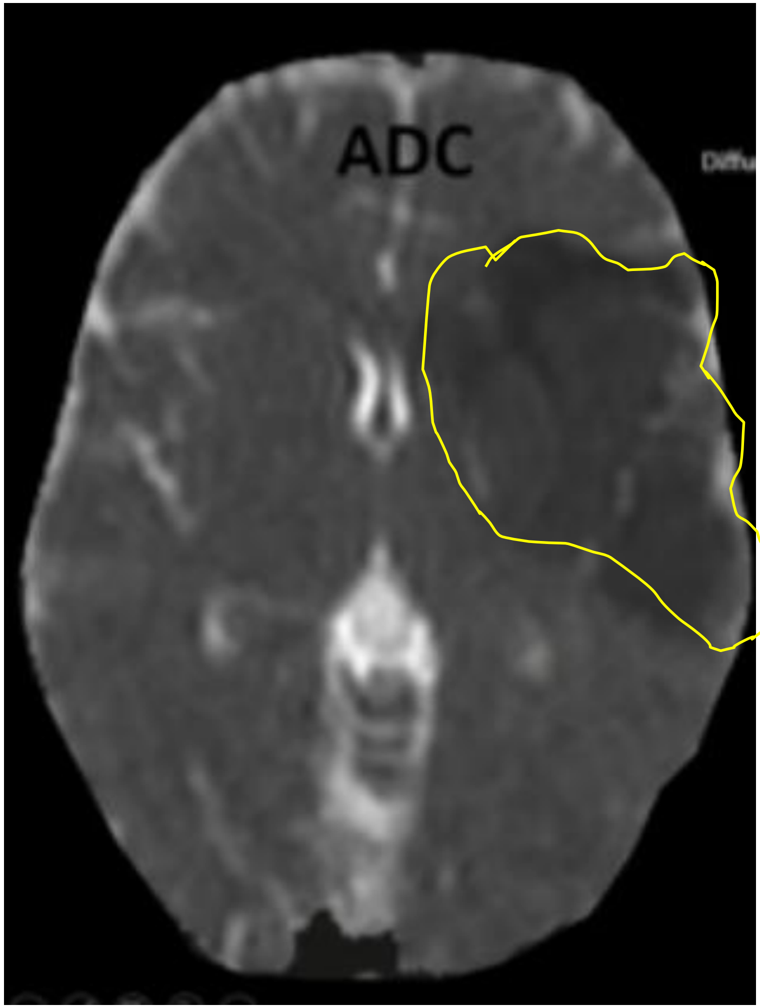
They appear basically as grayscale inverted DWI images.

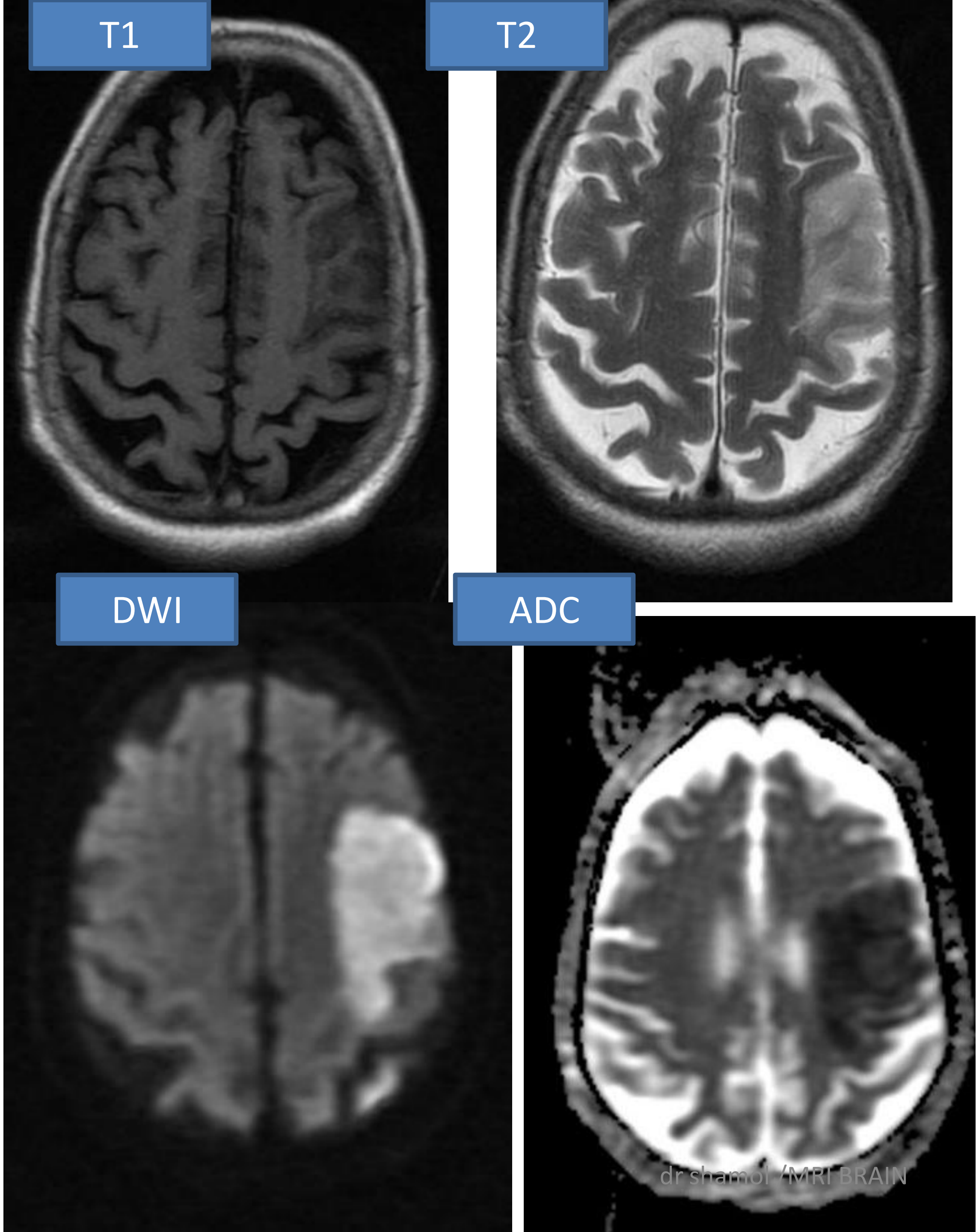
They are relatively low resolution image the following appearance:

grey matter:	intermediate signal intensity (grey)
white matter:	slightly hyperintense compared to grey matter
CSF:	high signal (white)
fat:	little signal due to paucity of water
other soft tissues	intermediate signal intensity (grey)

Acute pathology (ischaemic stroke, cellular tumour, pus)	usually appears as decreased signal (hypointense or black)denoting restricted diffusion.


DWI


ADC



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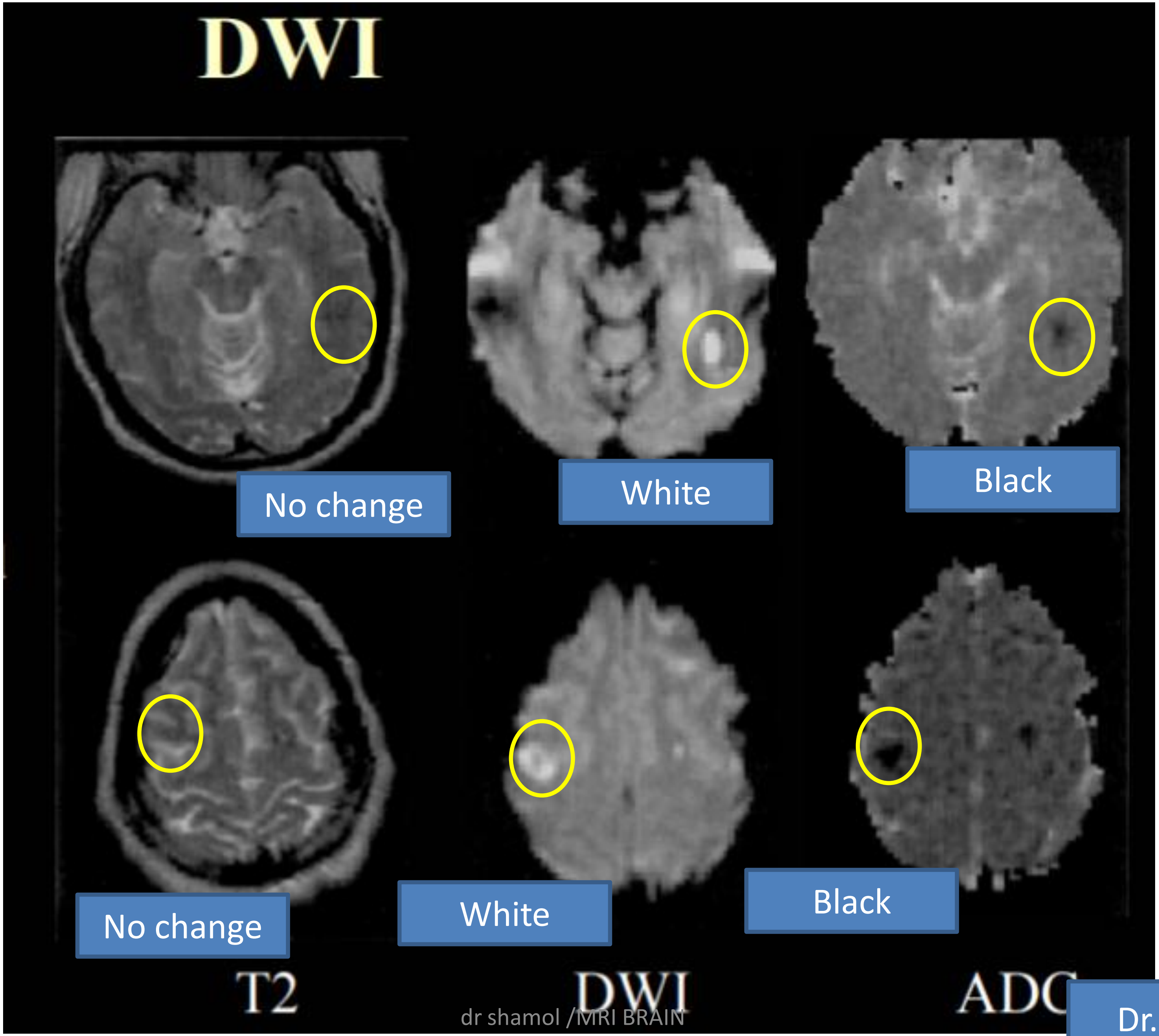
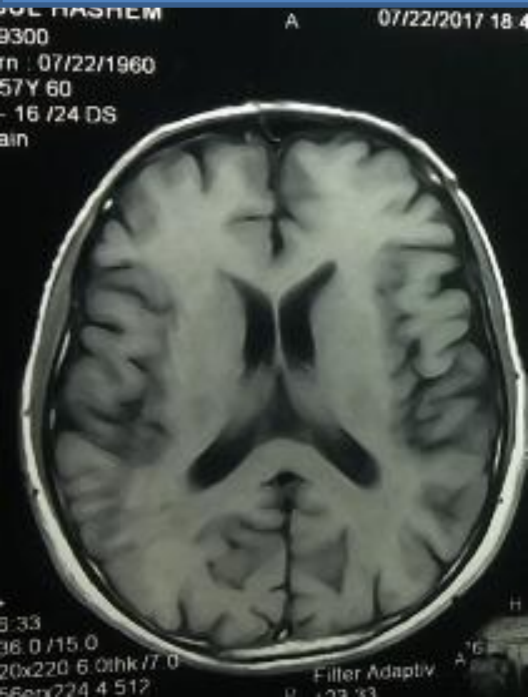
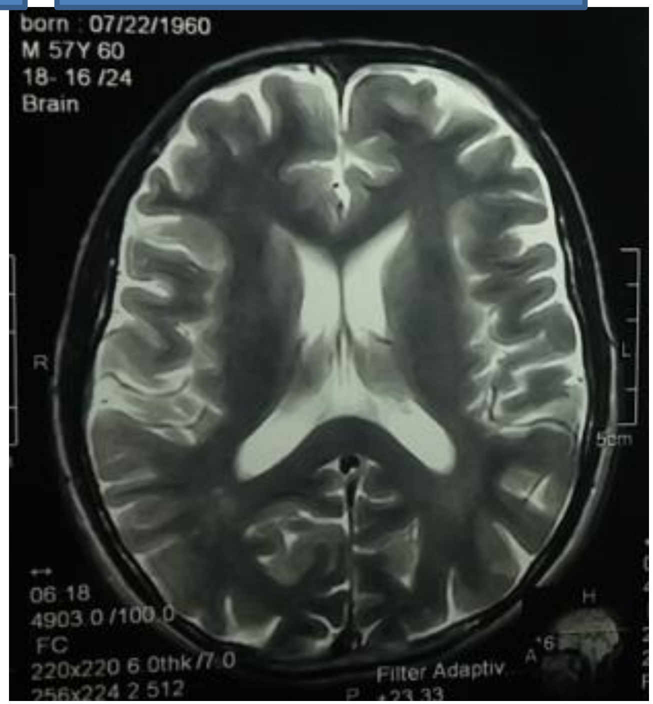

DWI
No change
White
Black
No change
White
Black
T2
DWI
ADC

AT GLANCE T1,T2, FLAIR

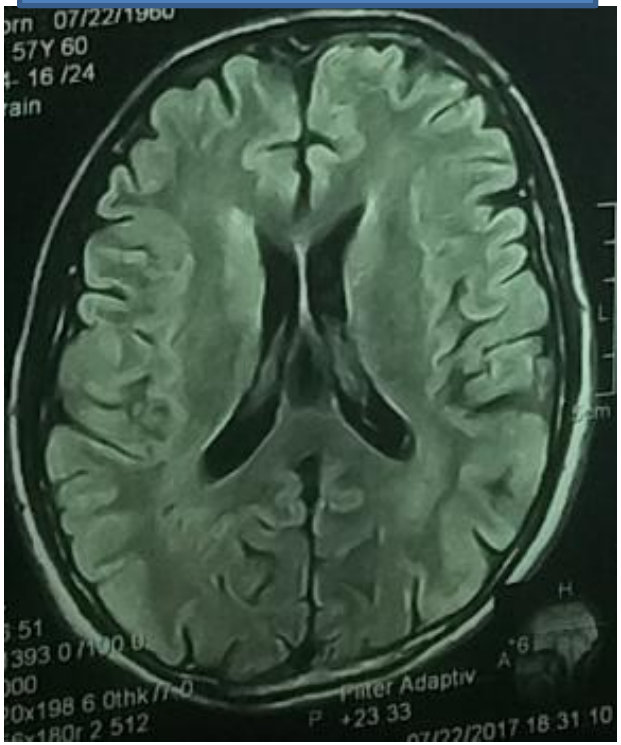
CSF– BLACK	CSF –WHITE	CSF-BLACK Border of ventricle is white



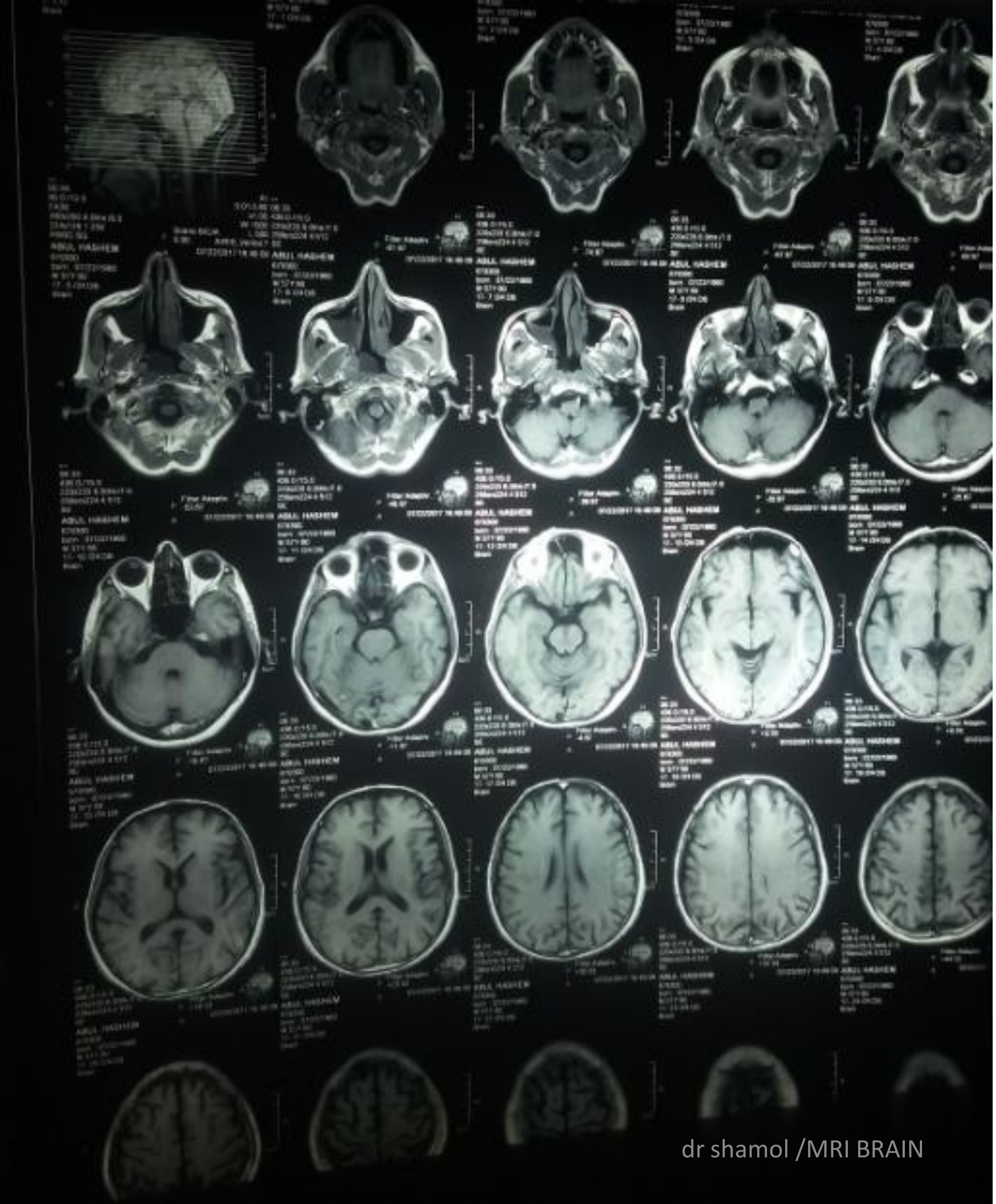
T1



T2

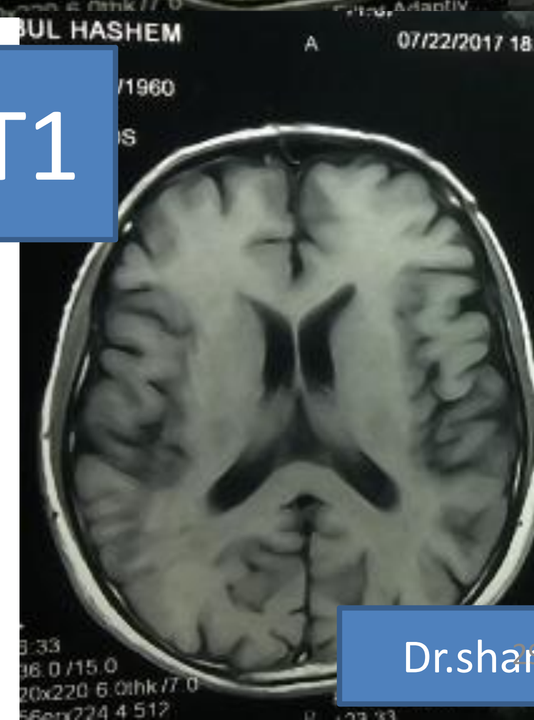


FLAIR

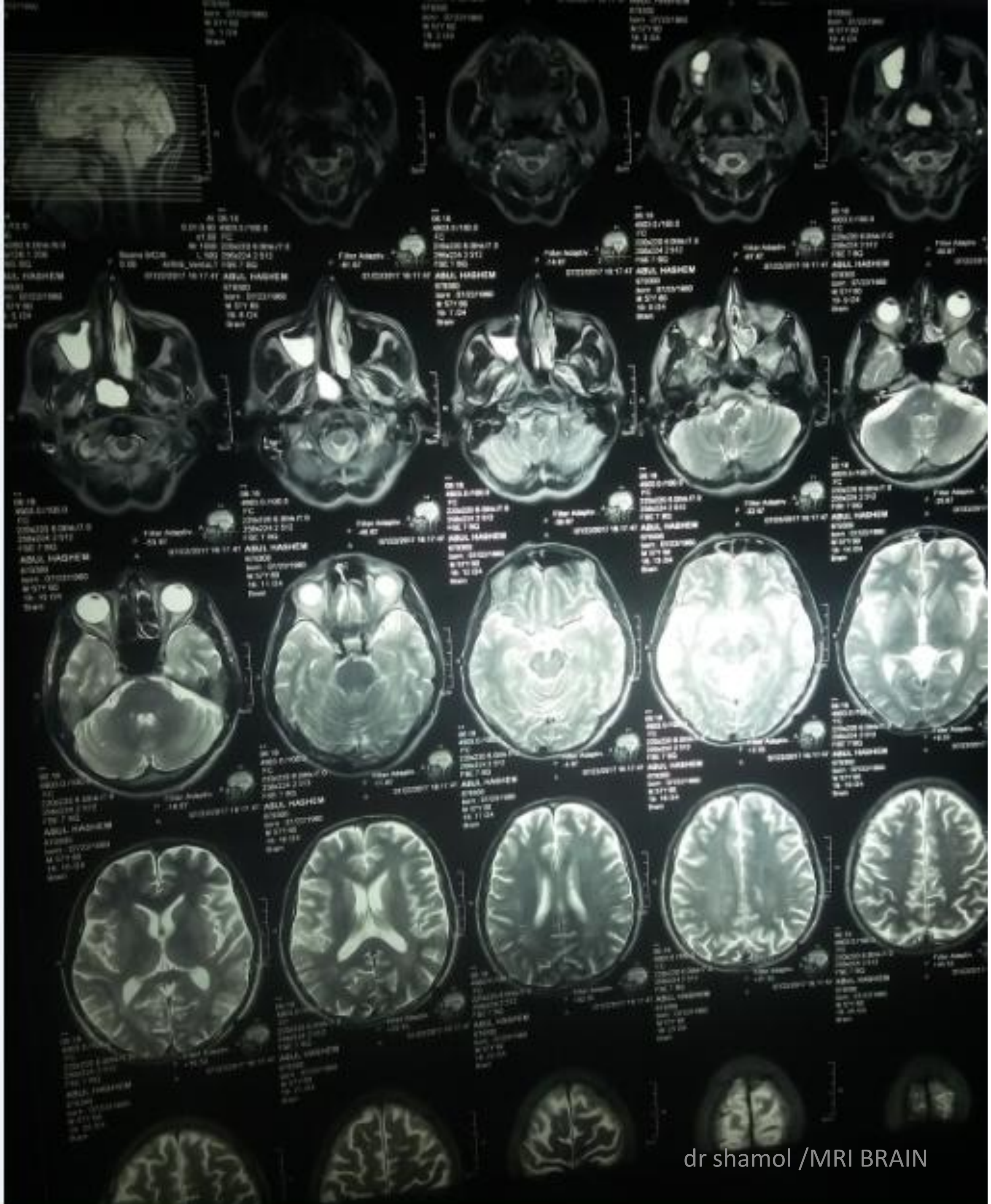


-18.67
L HASHEM
A
07/22/2017 18:49:09
07/22/1960
Y 60
15 /24 DS
6:33
36.0 /15.0

T1



UL HASHEM
A
07/22/2017 18:4
/1960
:33
6.0 /15.0
20x220 6.0thk /7.0
56en/224 4.512


dr shamol /MRI BRAIN


born : 07/22/1960
M 57Y 60
18- 16 /24
Brain
R
06 18
4903.0 /100.0
FC
220x220 6.0thk /7.0
Filter Adaptiv

T2


57Y 60
06 18
FC
256x224 2 512

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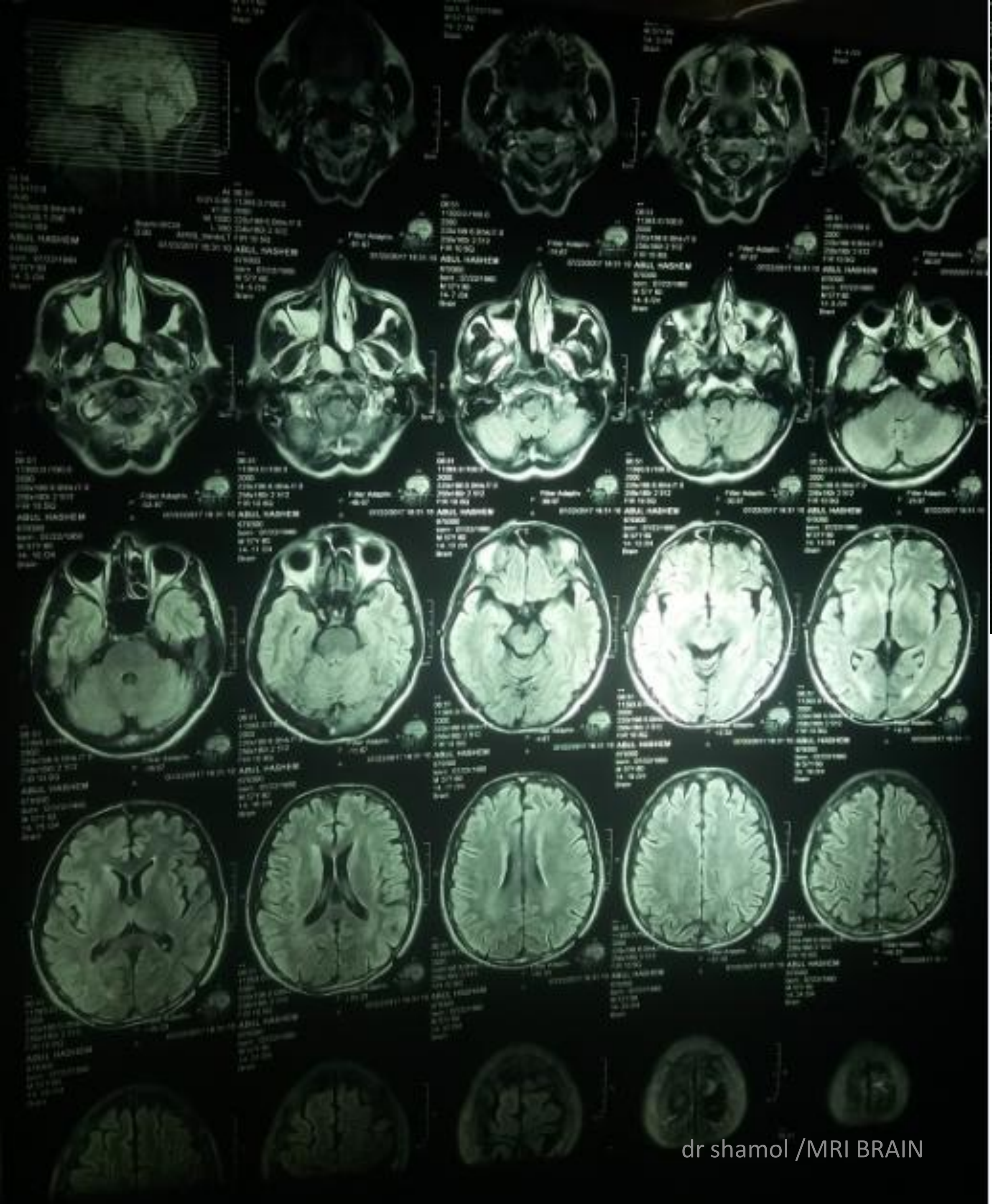
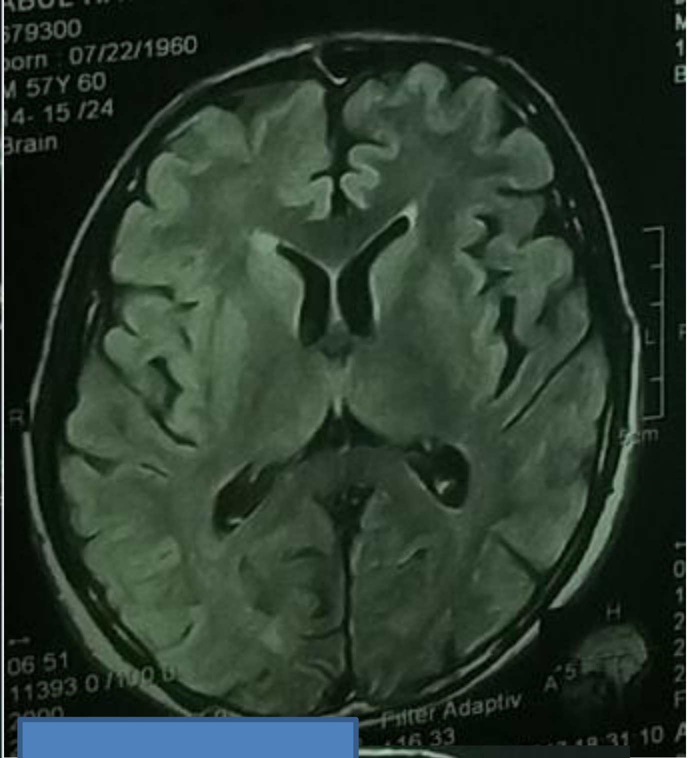
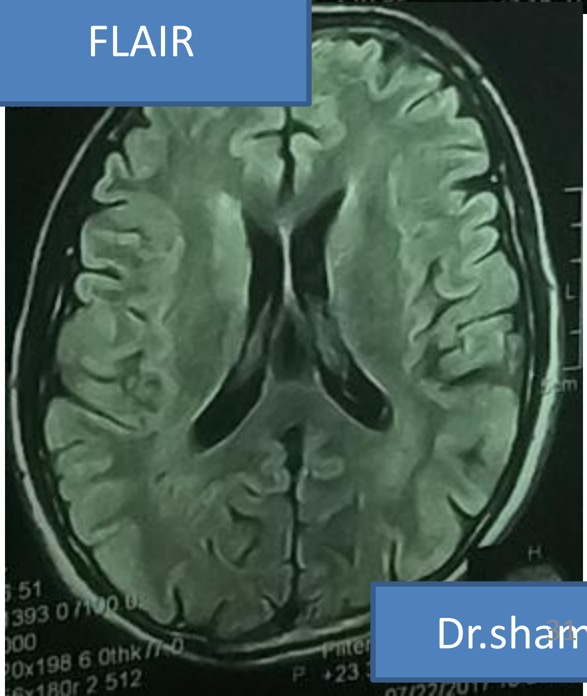




FLAIR



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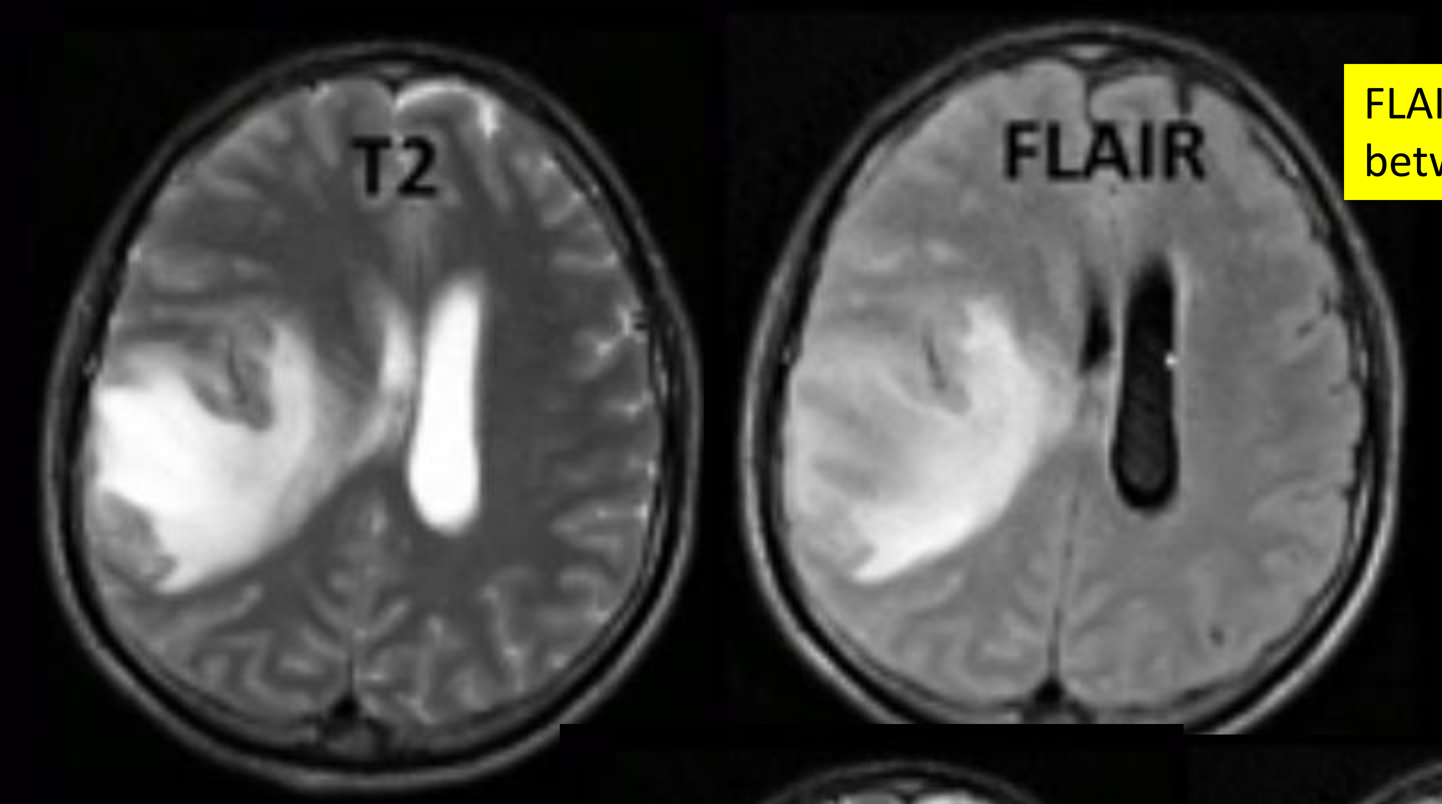
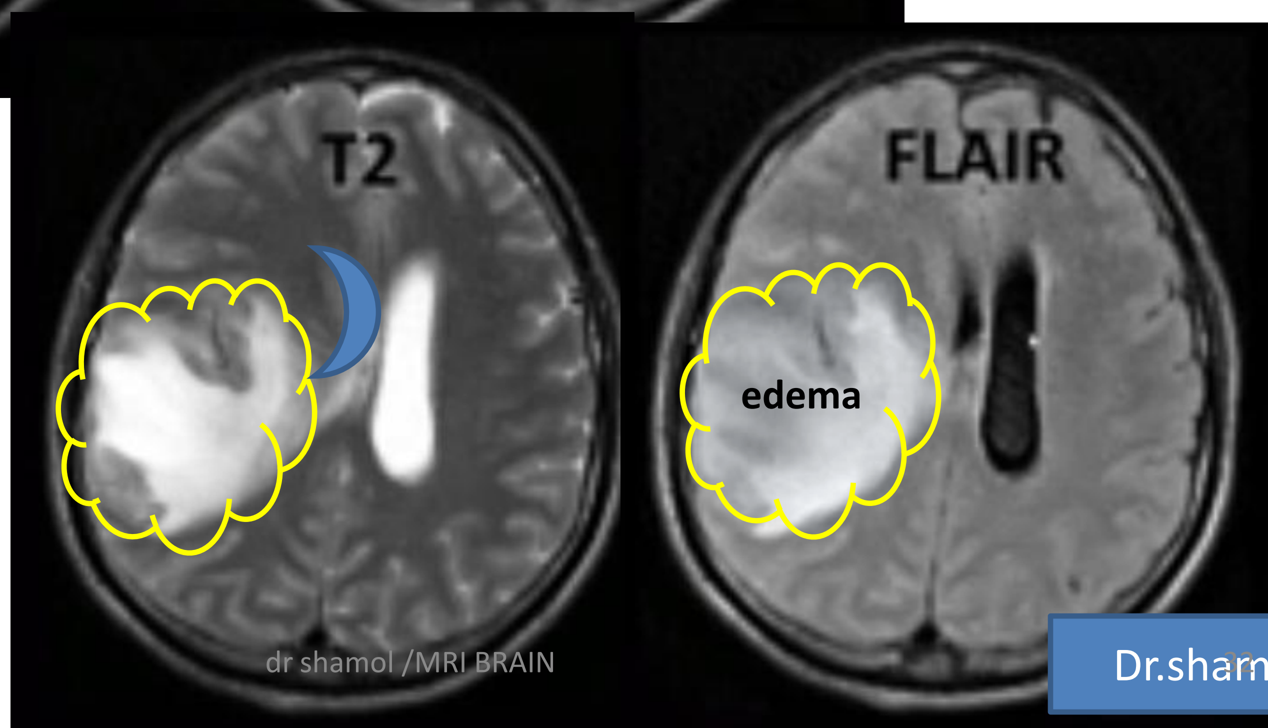
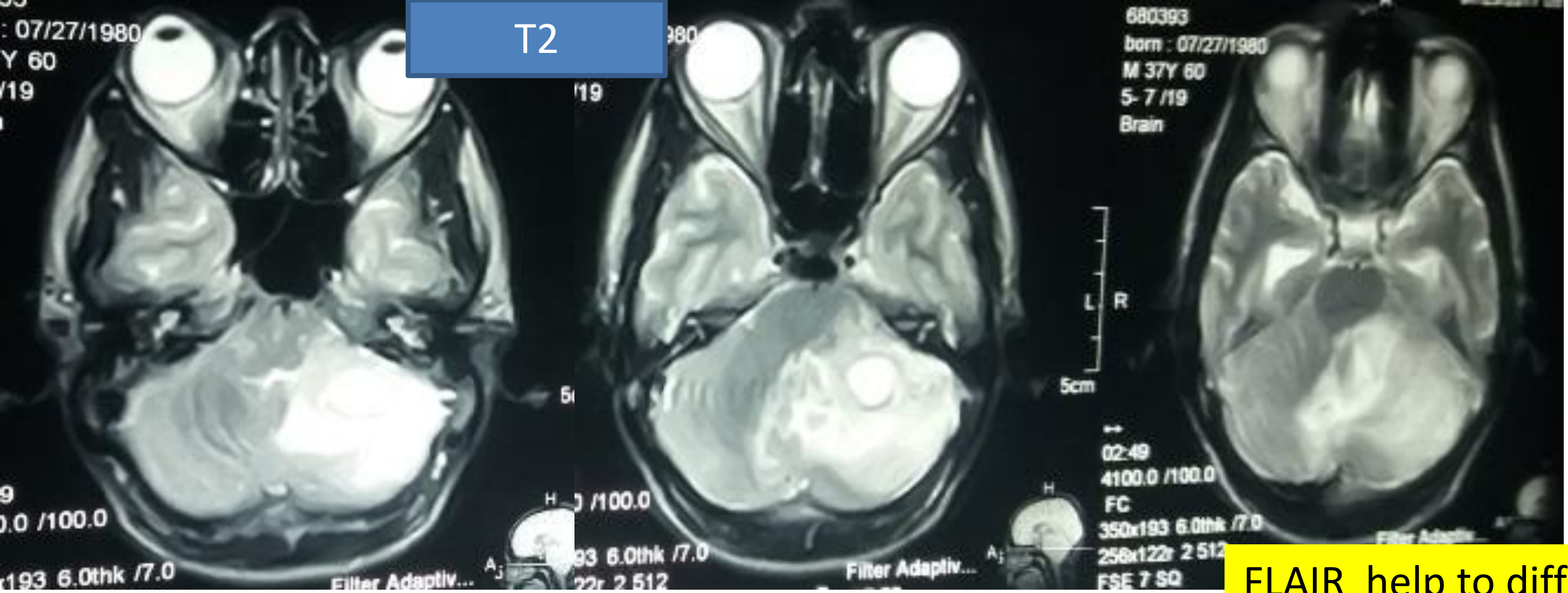
FLAIR help to differentiate between edema ventricle

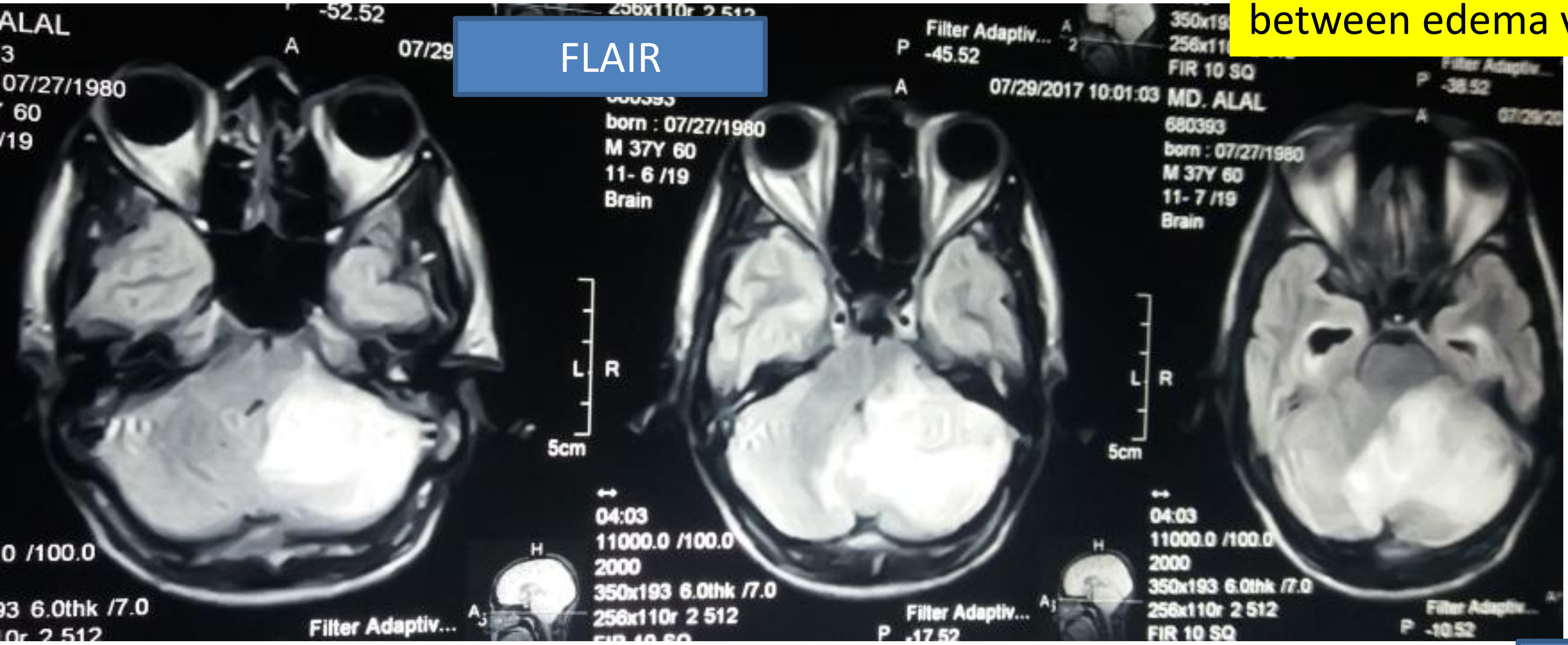




T2



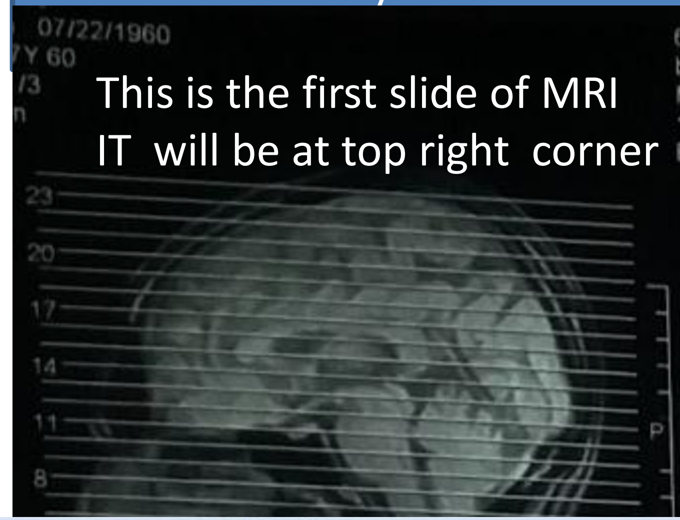
FLAIR



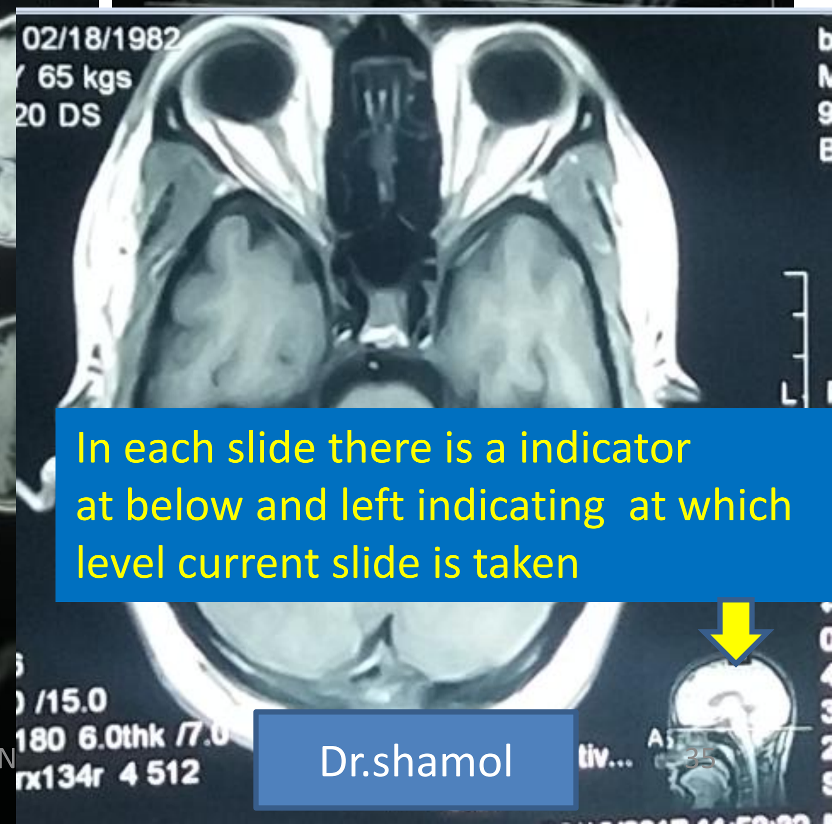
FLAIR help to differentiate between edema ventricle

HOW TO READ A MRI FILM
The structure you needed to indentify

How to indentify axial MRI FILM



This is the first slide of MRI
IT will be at top right corner



In each slide there is a indicator
at below and left indicating at which
level current slide is taken

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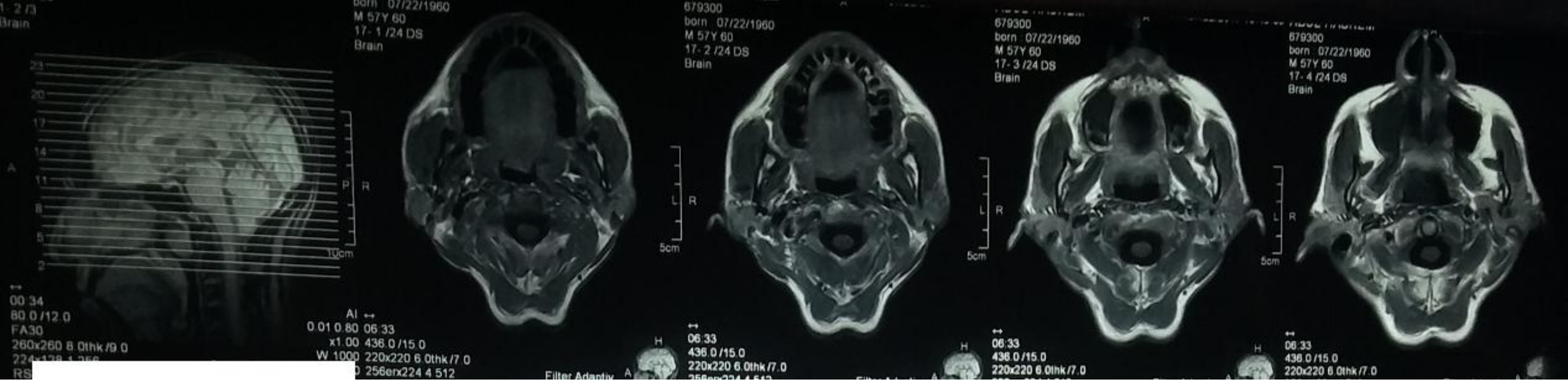
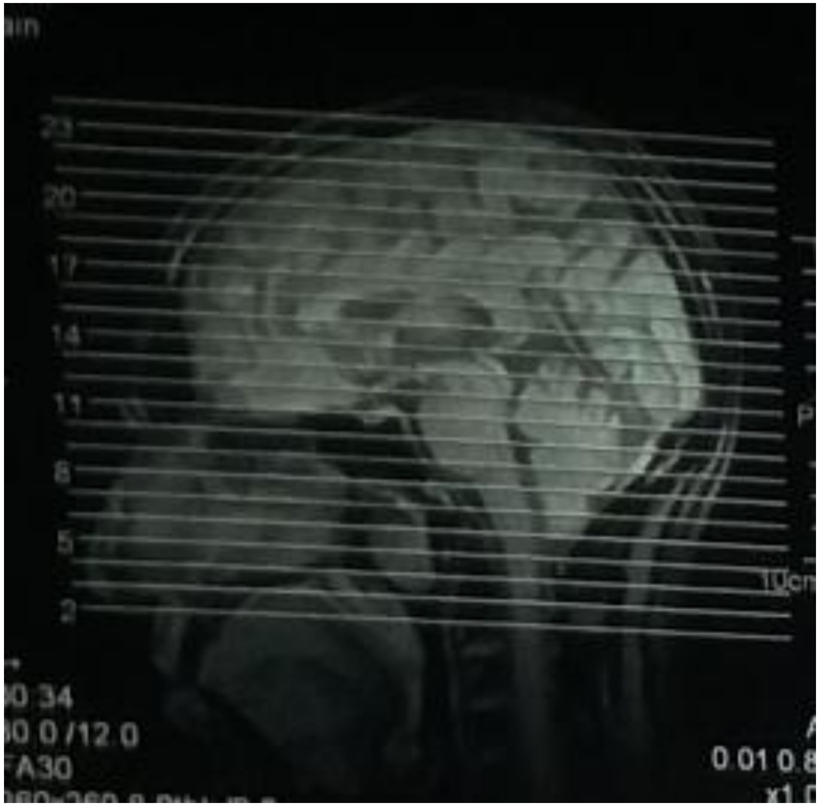
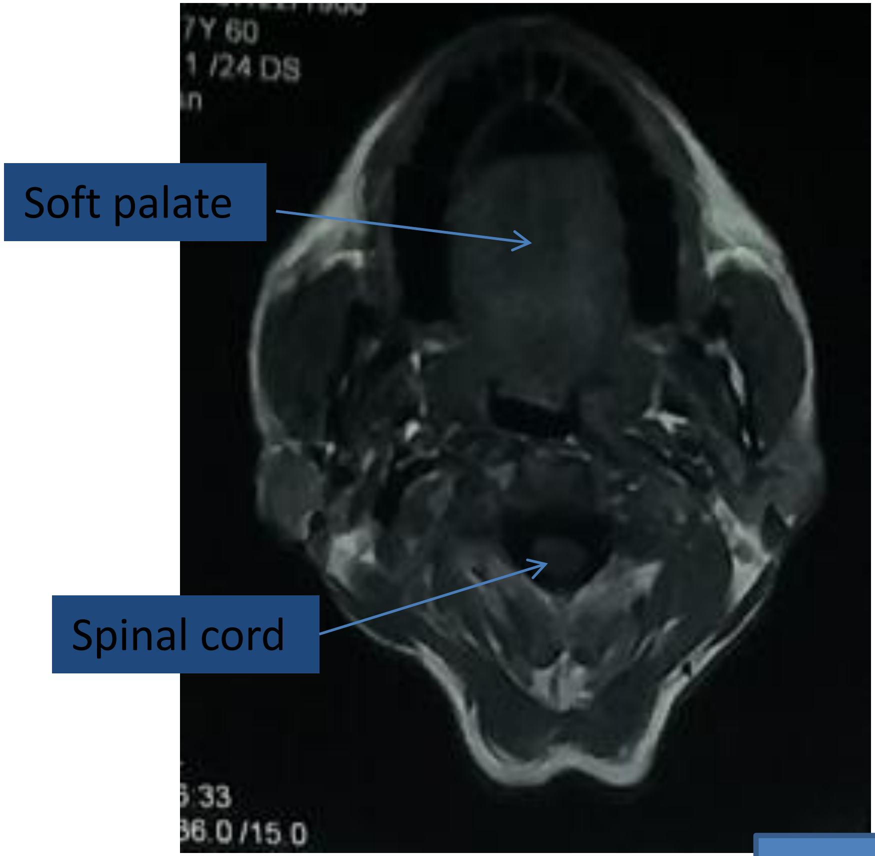
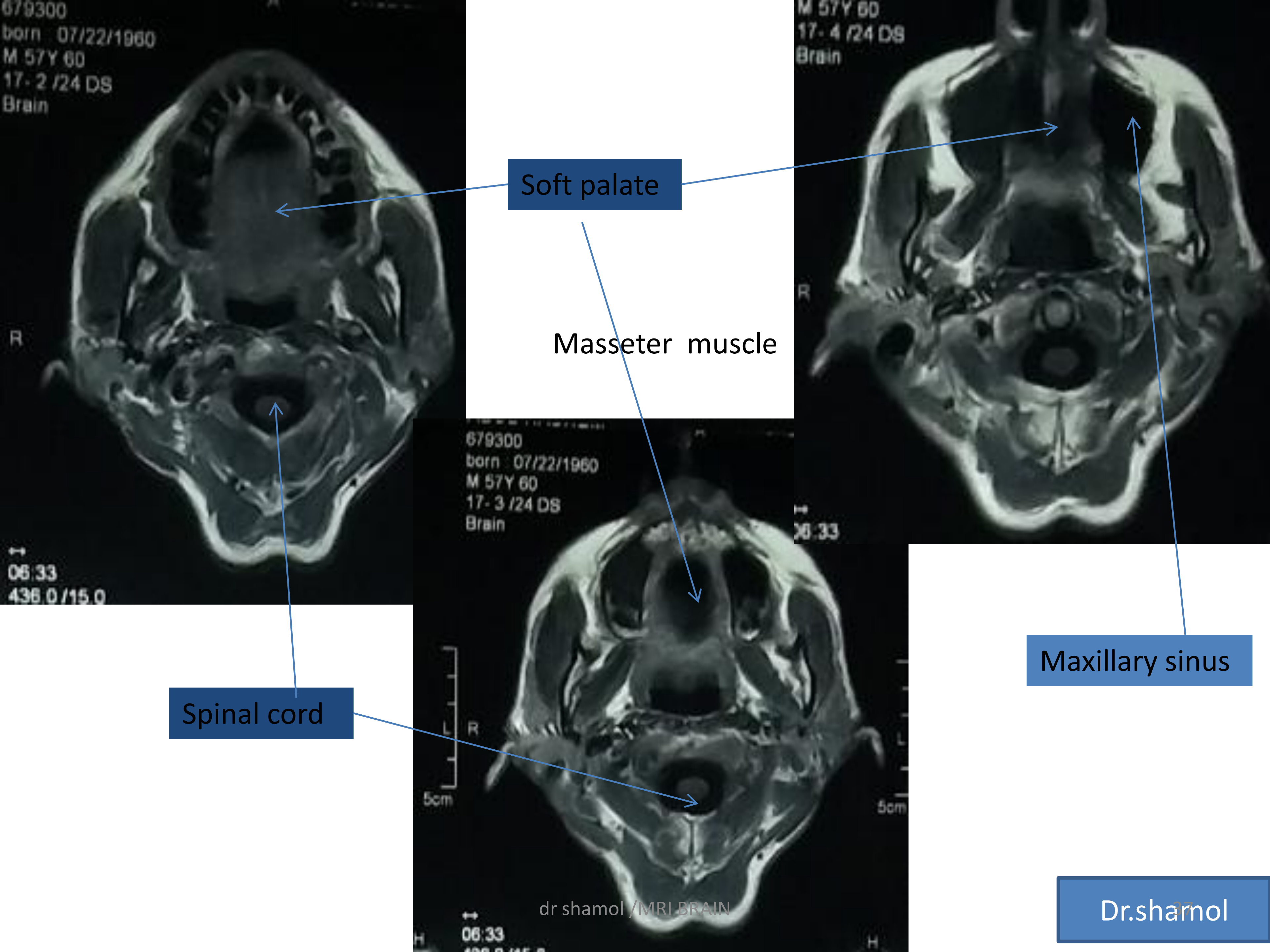
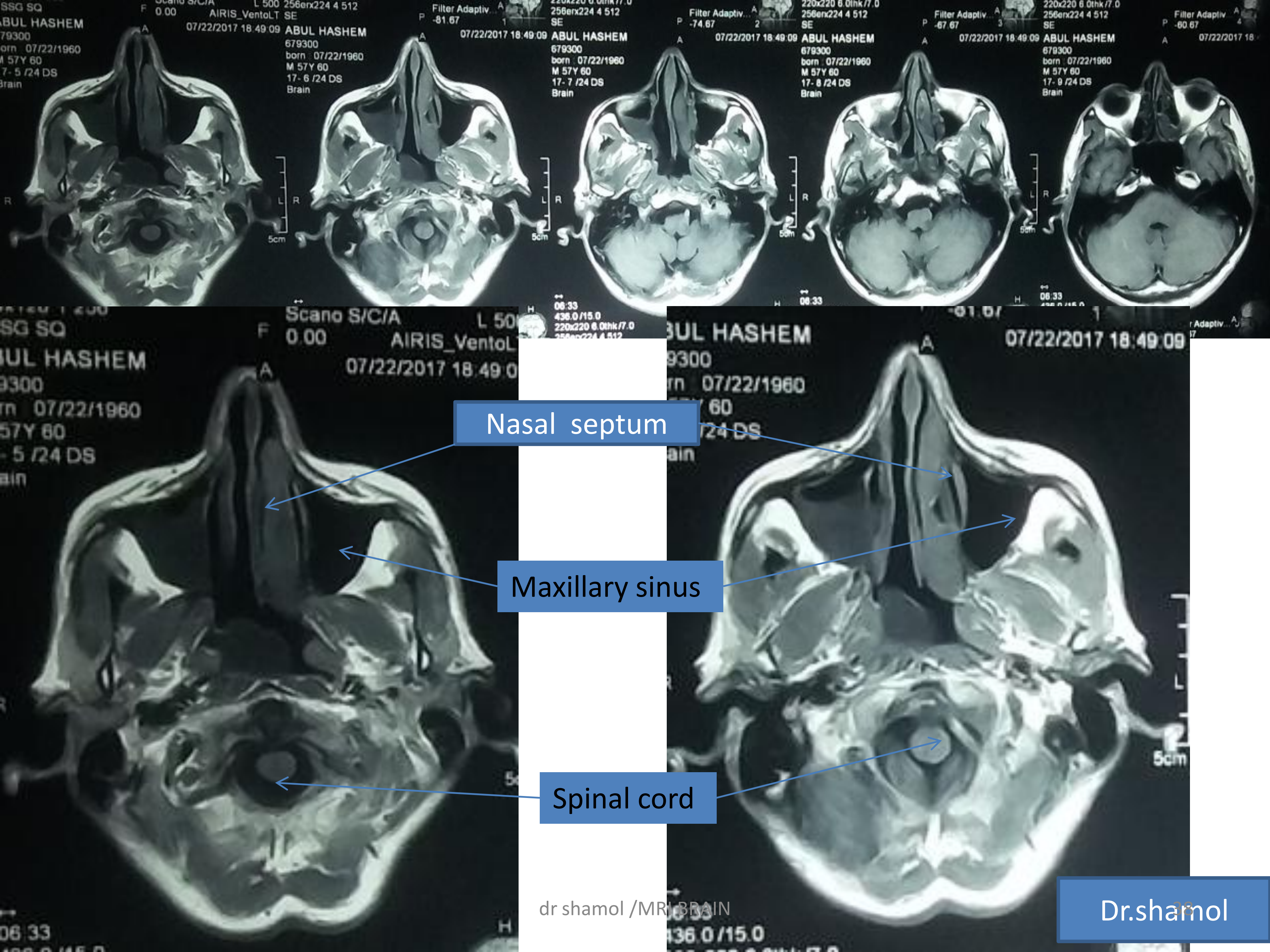
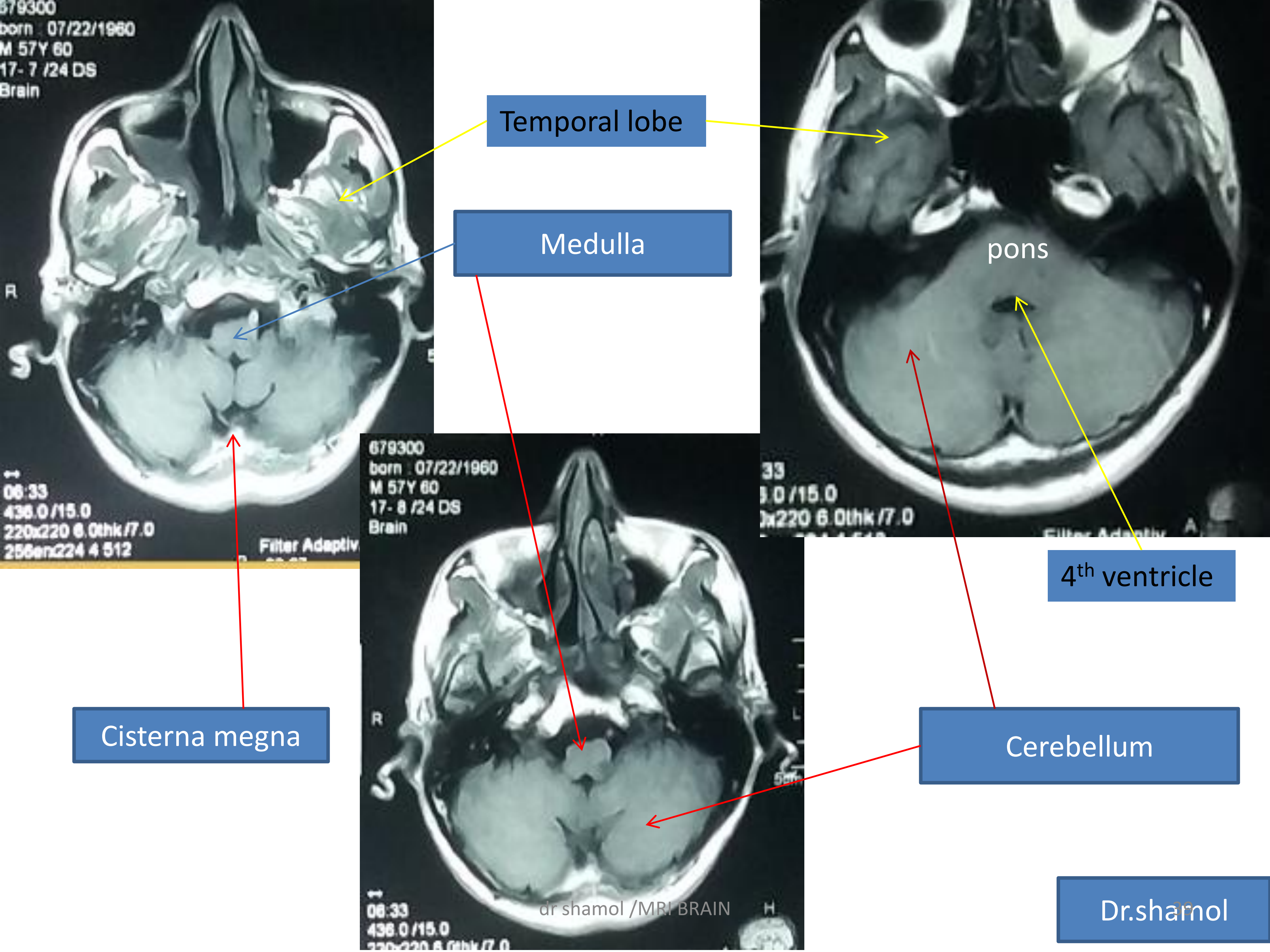

679300
born 07/22/1960
M 57Y 60
Brain
5cm
10cm

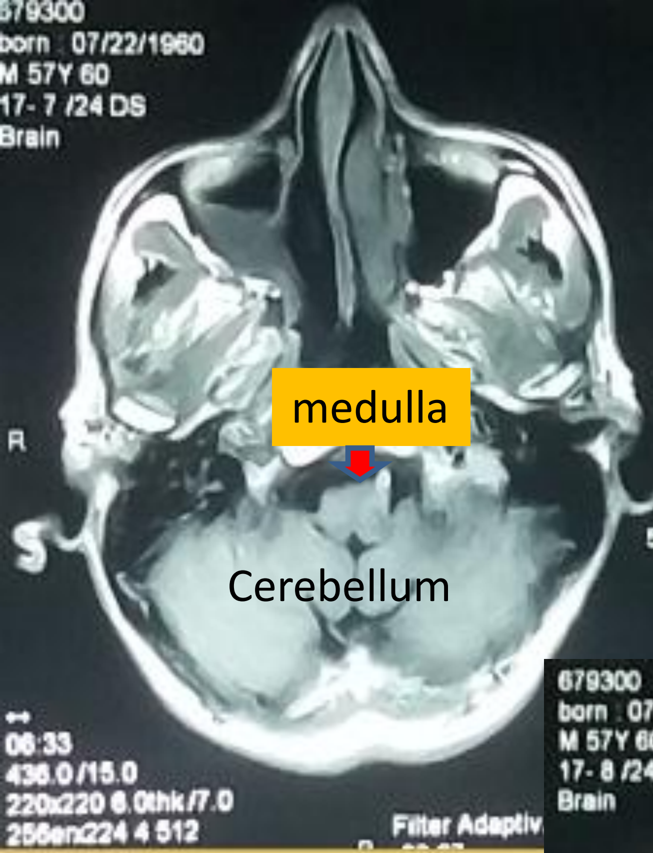


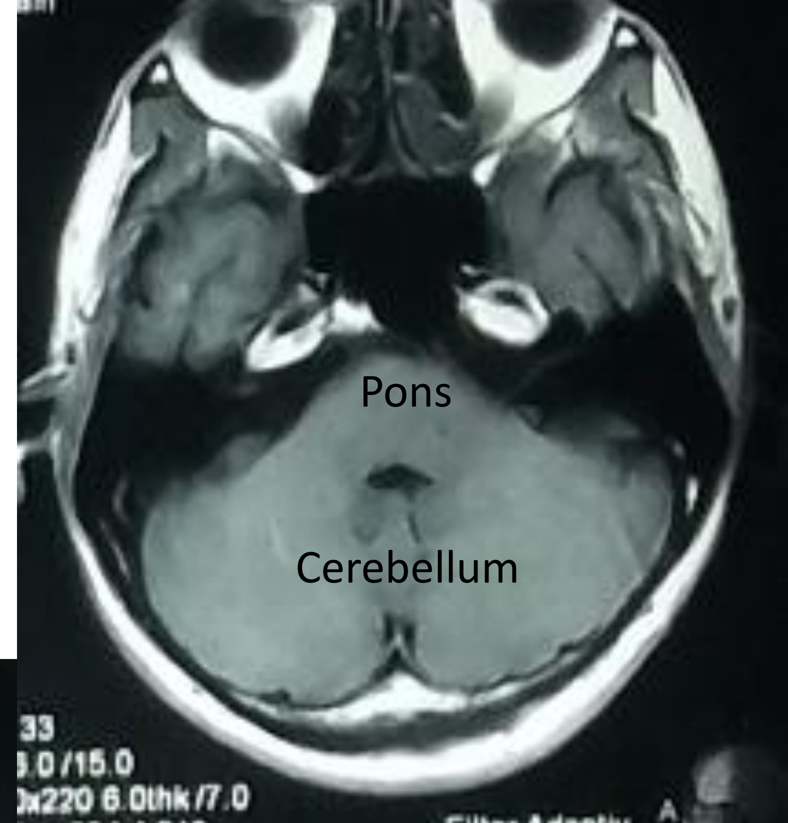

Soft palate
Spinal cord

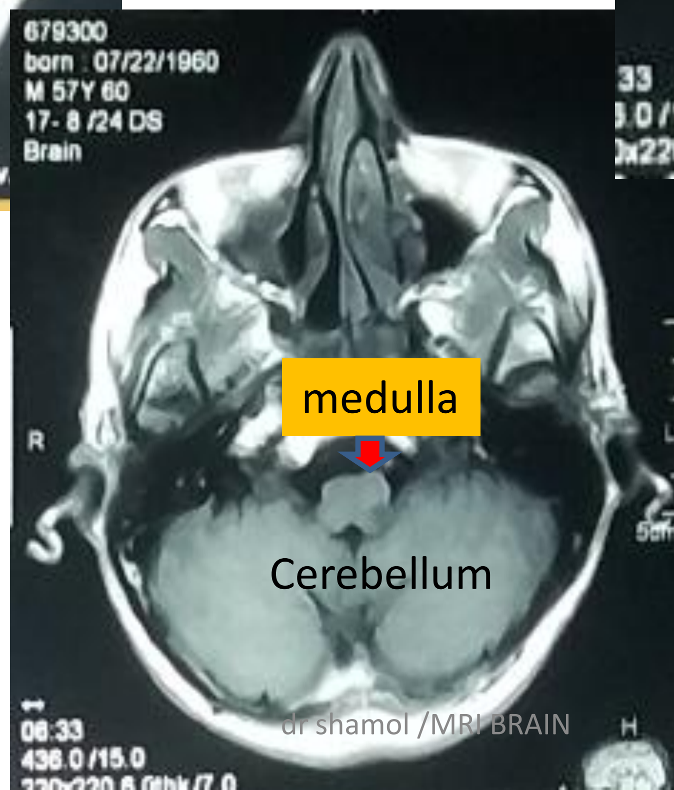

Soft palate
Masseter muscle
Maxillary sinus
Spinal cord
679300
born 07/22/1960
M 57Y 60
17- 2 /24 DS
Brain
R
06:33
436.0 /15.0
M 57Y 60
17- 4 /24 DS
Brain
R
06:33
679300
born 07/22/1960
M 57Y 60
17- 3 /24 DS
Brain
R
L
5cm
5cm
06:33
Dr.shamol


Nasal septum
Maxillary sinus
Spinal cord
Dr.shamol


Temporal lobe
Medulla
pons
4th ventricle
Cisterna megna
Cerebellum
Dr.shamol

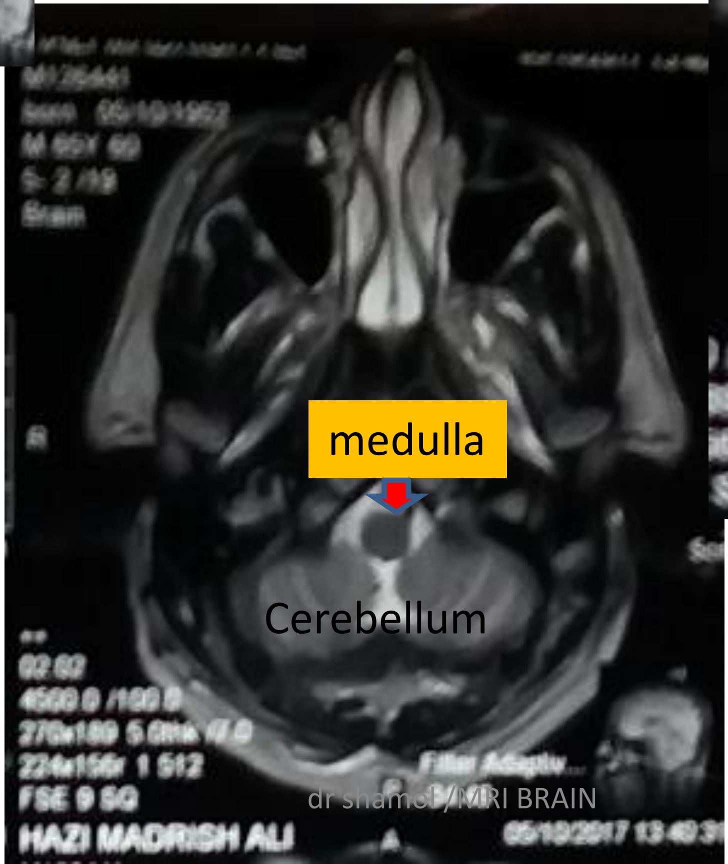

679300
born : 07/22/1960
M 57Y 60
17- 7 /24 DS
Brain
R
medulla
Cerebellum
06:33
436.0 /15.0
220x220 6.0thk /7.0
256en224 4 512
Filter Adaptiv

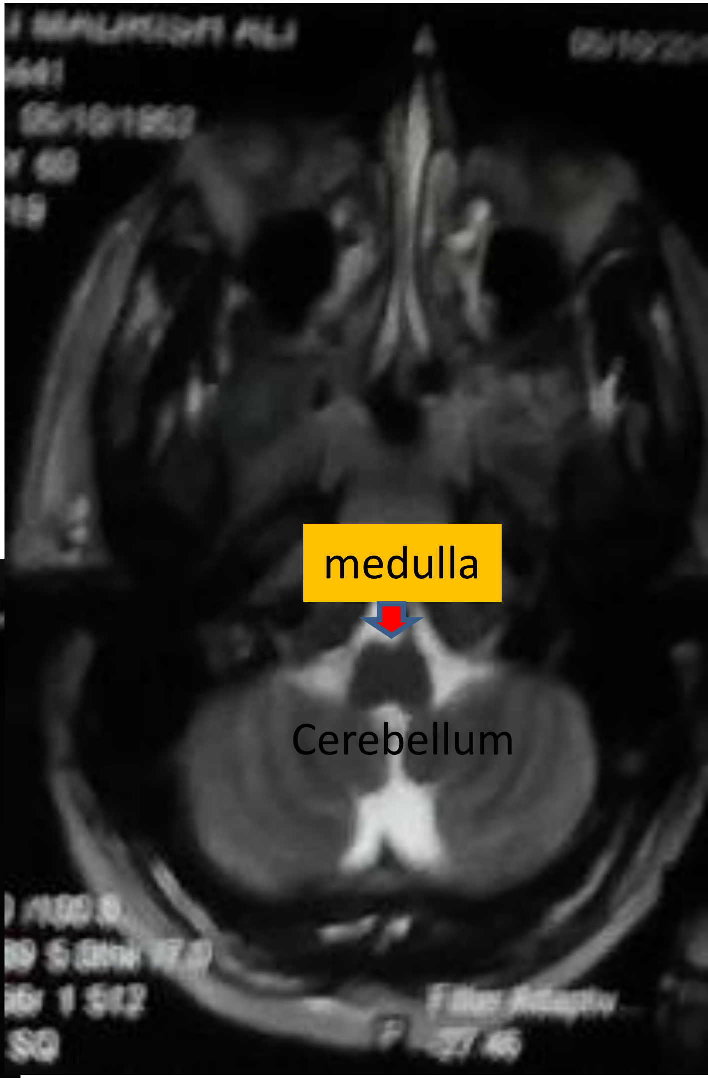

Pons
Cerebellum
33
Filter Adaptiv


679300
born : 07/22/1960
M 57Y 60
17- 8 /24 DS
Brain
R
medulla
Cerebellum
06:33
436.0 /15.0

T1

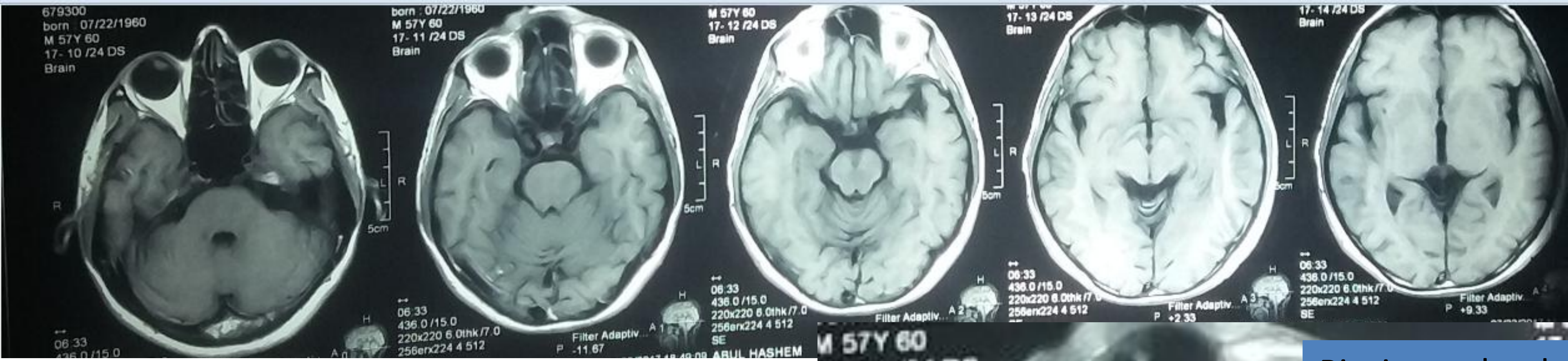
Dr.shamol



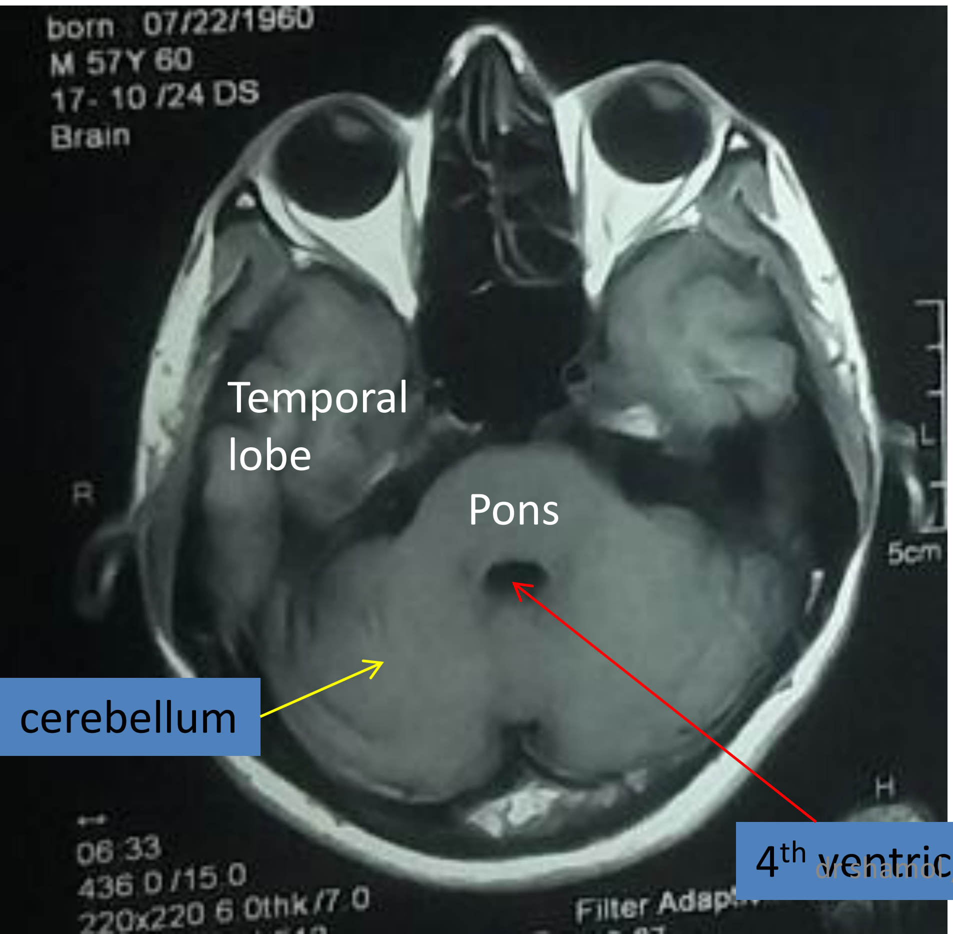


T2

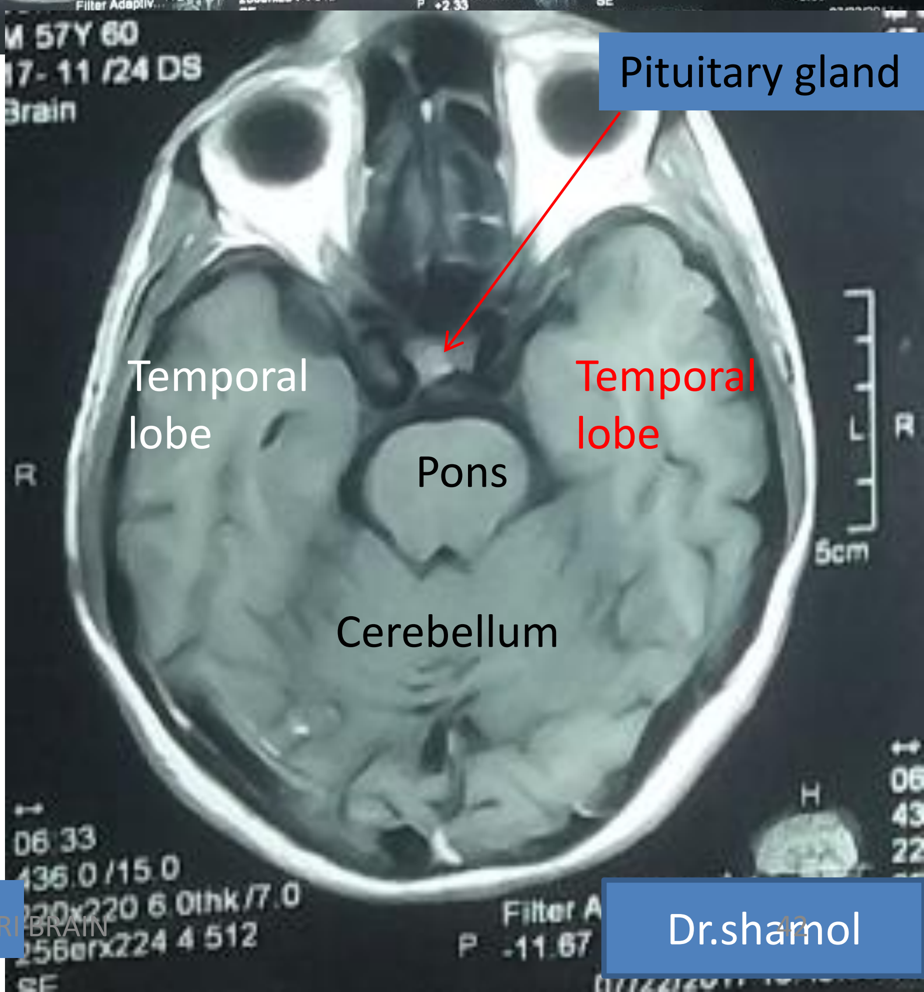
Dr.shamol



born 07/22/1960
M 57Y 60
17- 10 /24 DS
Brain
17- 11 /24 DS
17- 12 /24 DS
17- 13 /24 DS
17- 14 /24 DS
5cm
ABUL HASHEM

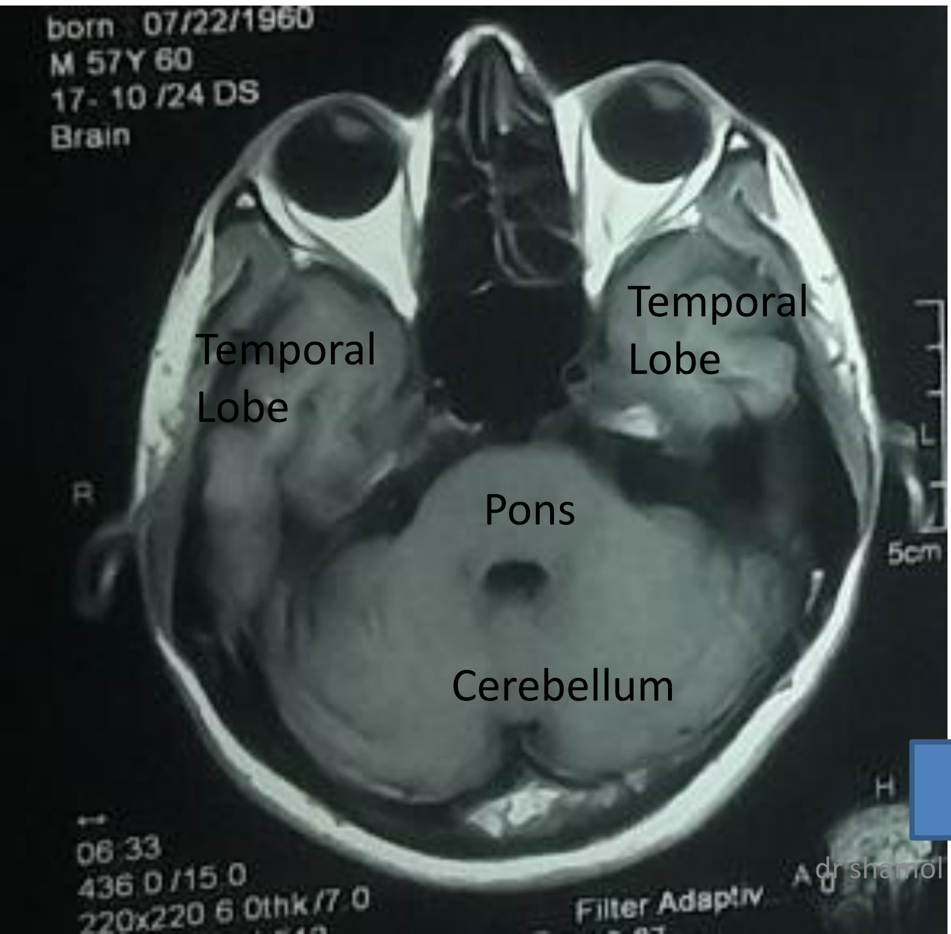


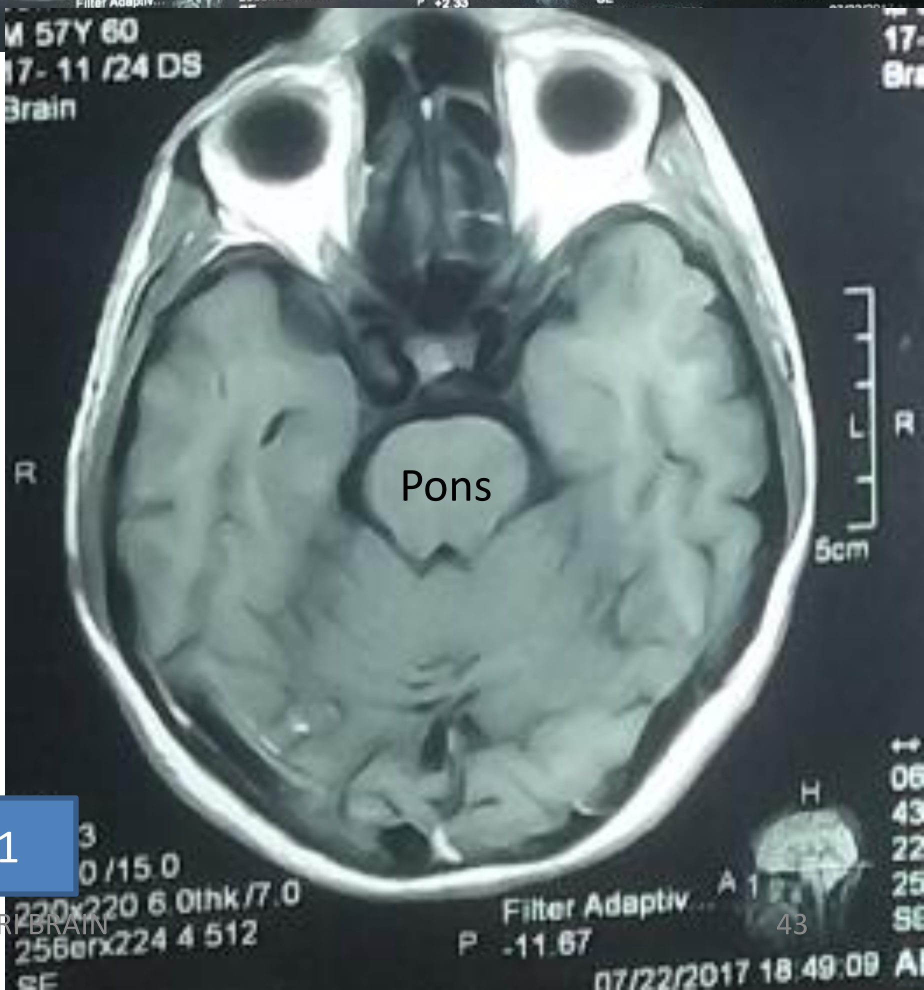
born 07/22/1960
M 57Y 60
17- 10 /24 DS
Brain
Temporal lobe
Pons
cerebellum
4th ventricle
5cm
06:33
436.0 /15.0
220x220 6.0thk /7.0



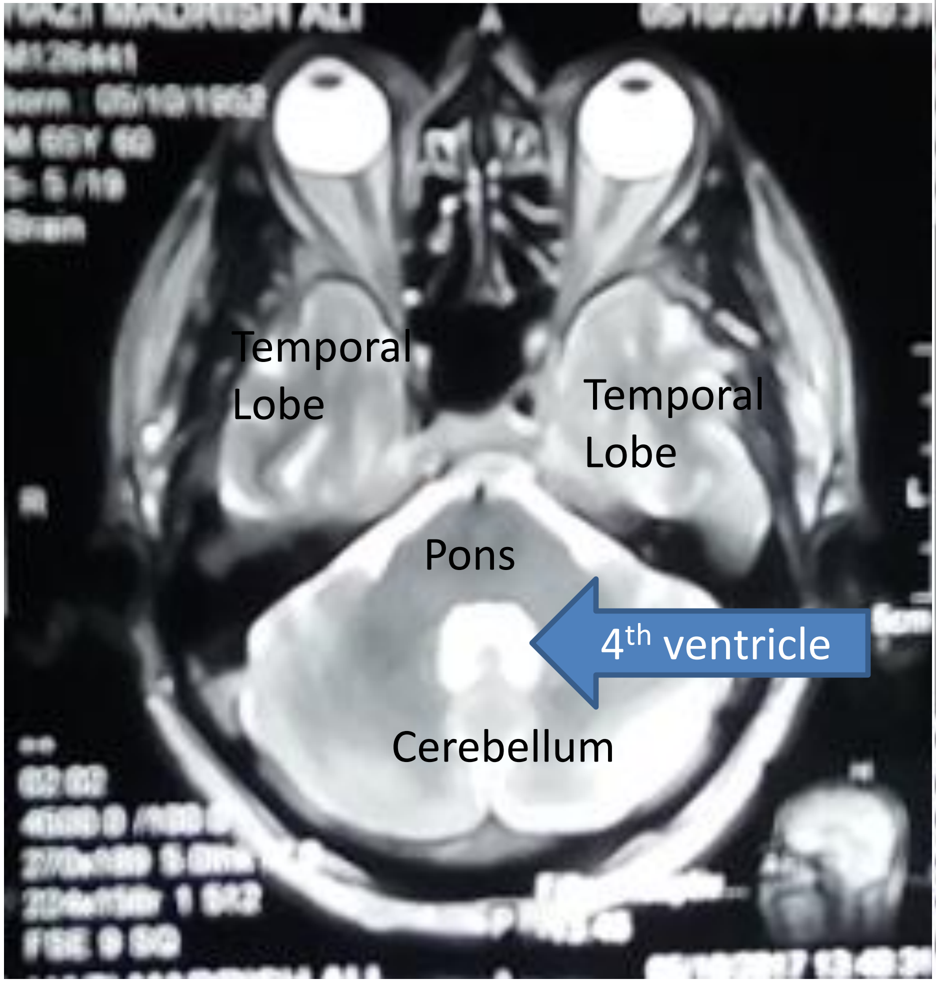
M 57Y 60
17- 11 /24 DS
Brain
Pituitary gland
Temporal lobe
Temporal lobe
Pons
Cerebellum
5cm
06:33
436.0 /15.0
220x220 6.0thk /7.0
256erx224 4 512
P -11.67

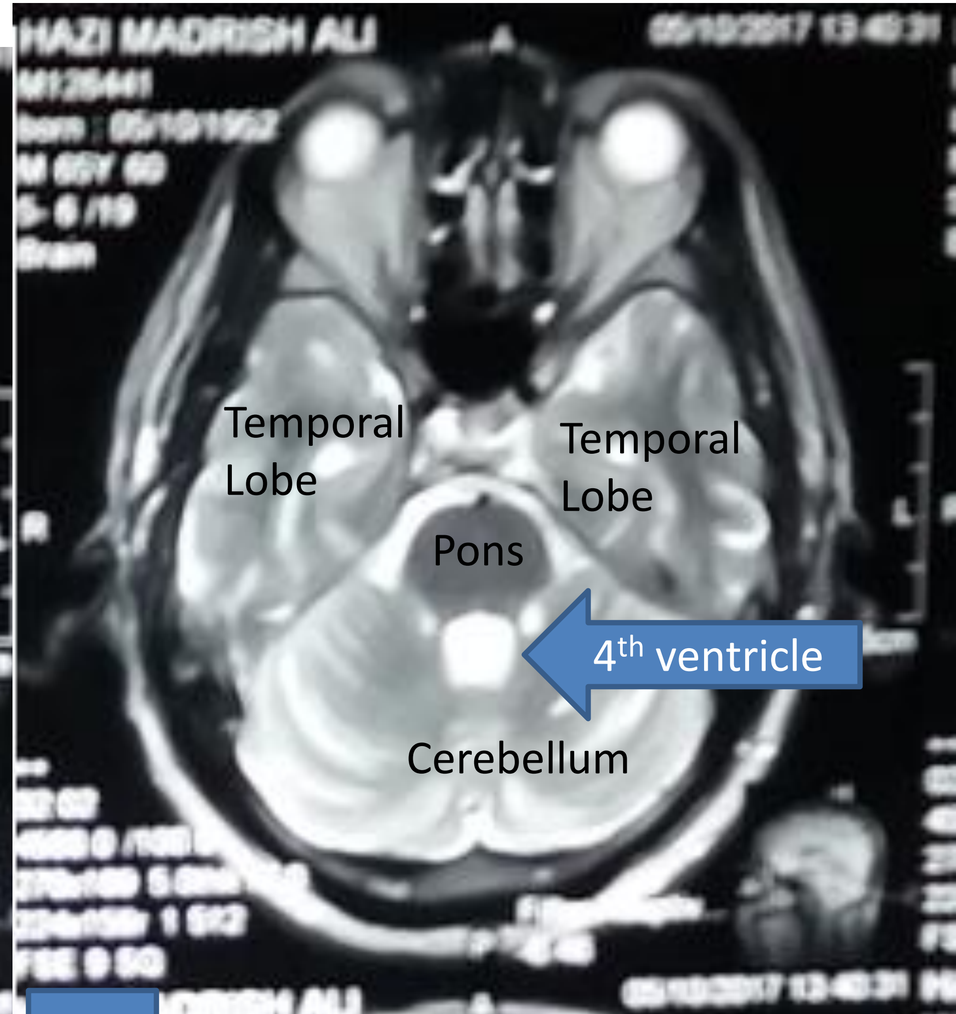
Dr.shamol


born 07/22/1960
M 57Y 60
17- 10 /24 DS
Brain
Temporal Lobe
Temporal Lobe
Pons
Cerebellum
5cm
R
06:33
436.0 /15.0
220x220 6.0thk /7.0
Filter Adaptiv


M 57Y 60
17- 11 /24 DS
Brain
Pons
R
5cm
0 /15.0
220x220 6.0thk /7.0
256erx224 4 512
Filter Adaptiv
P -11.67
07/22/2017 18:49:09

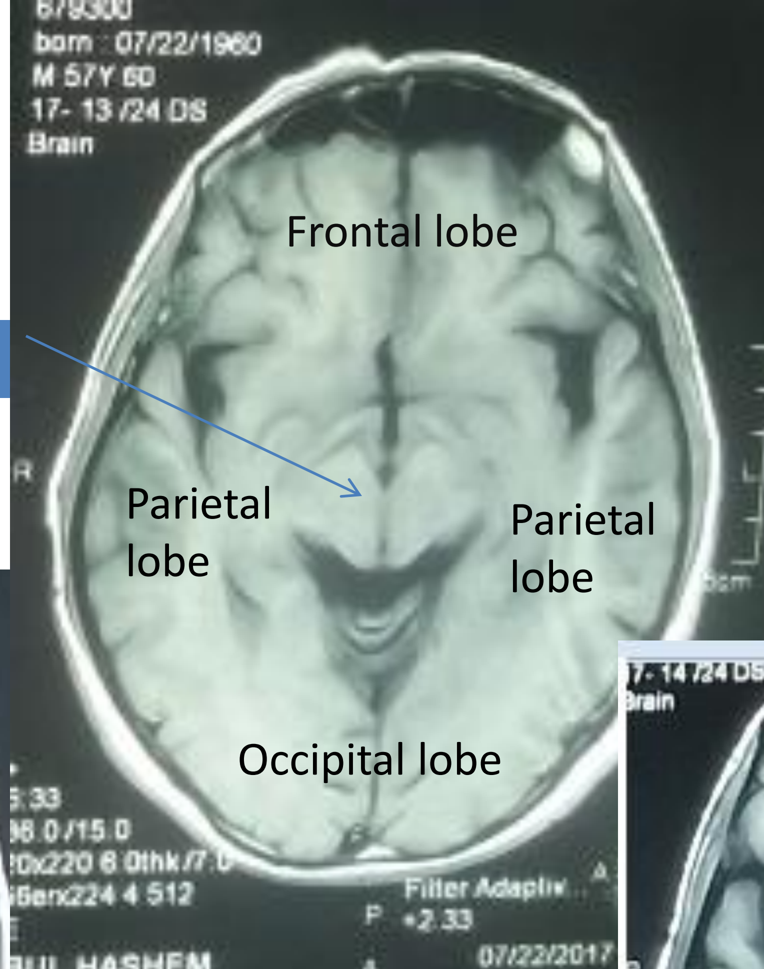
T1

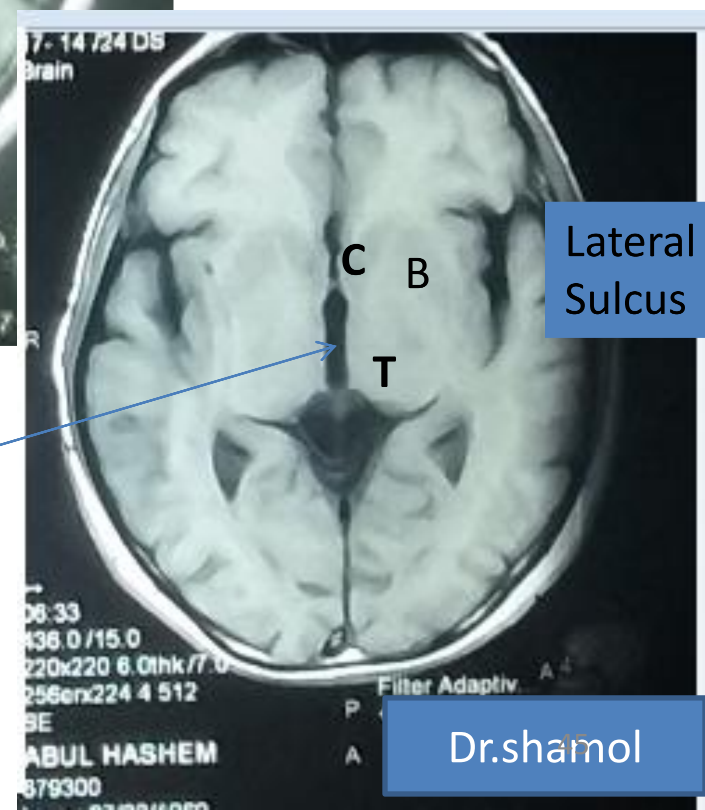

Temporal
Lobe
Temporal
Lobe
Pons
4th ventricle
Cerebellum

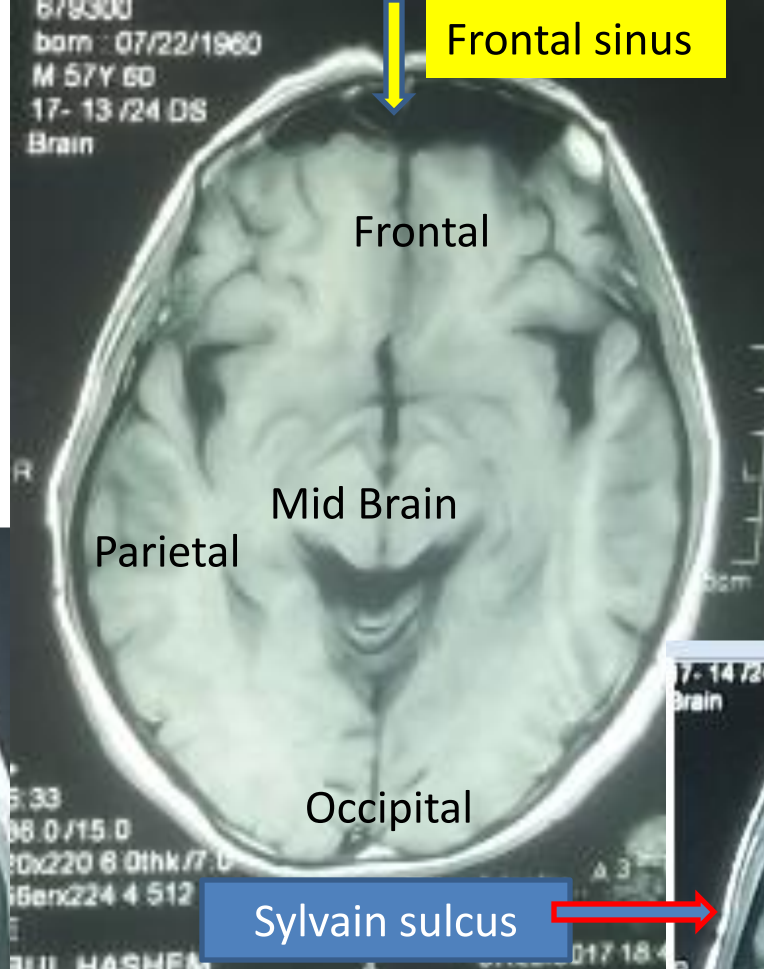

Temporal
Lobe
Temporal
Lobe
Pons
4th ventricle
Cerebellum

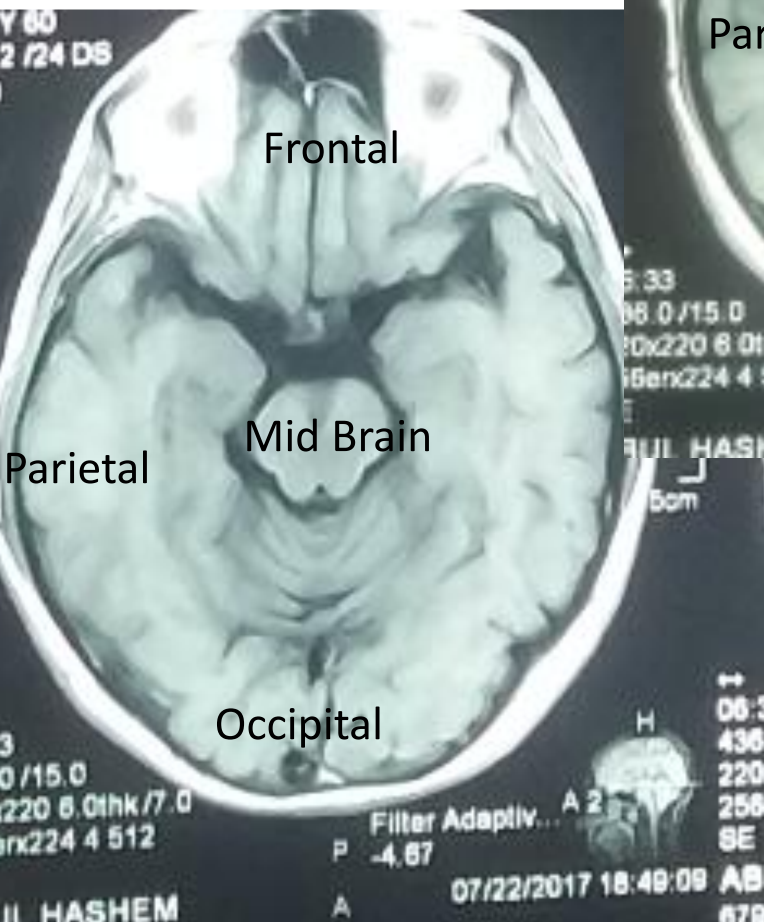
T2

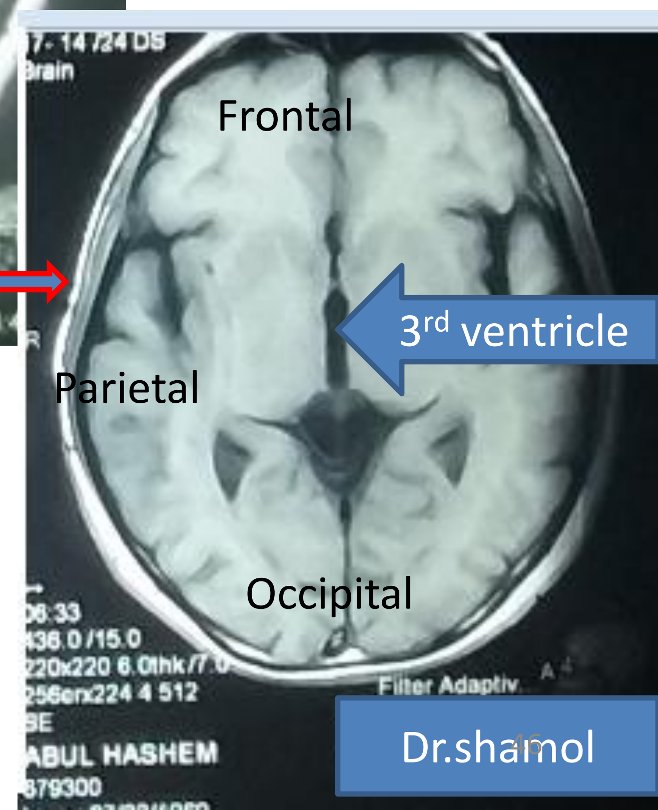
C-caudate nucleus
B-basal ganglia
T-thalamus

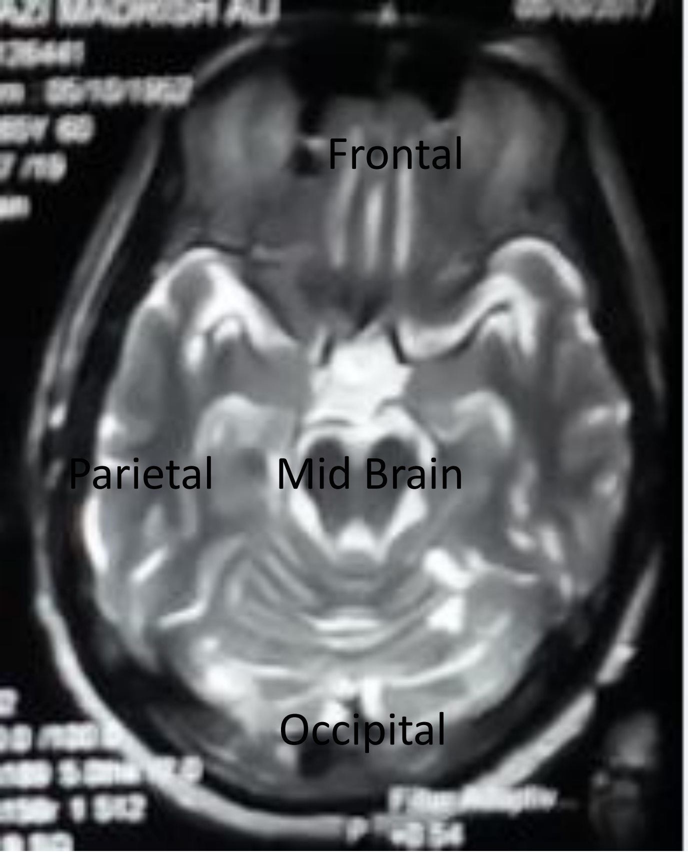


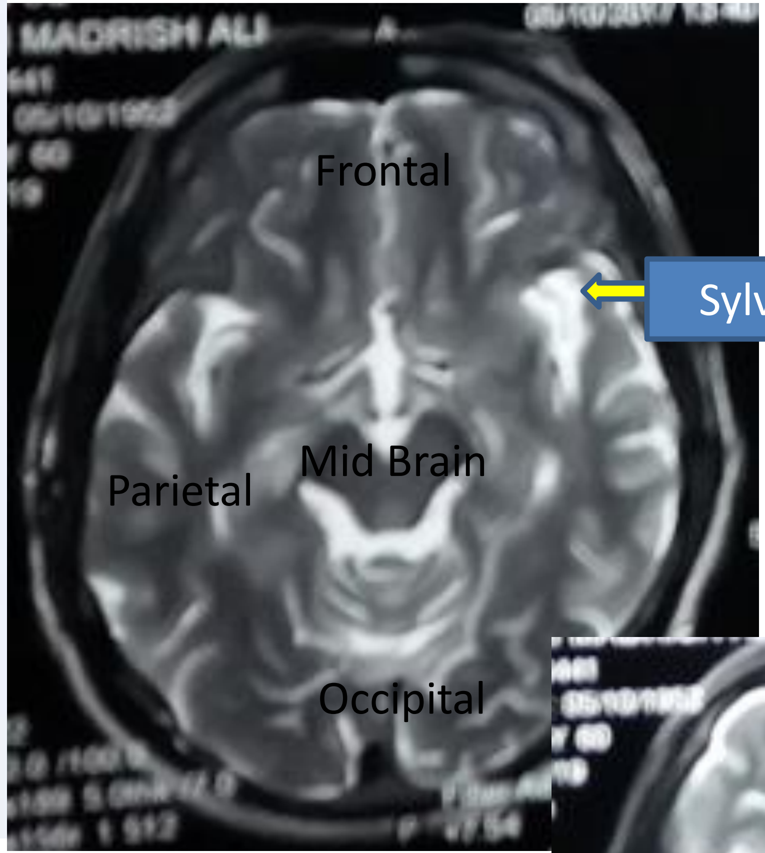


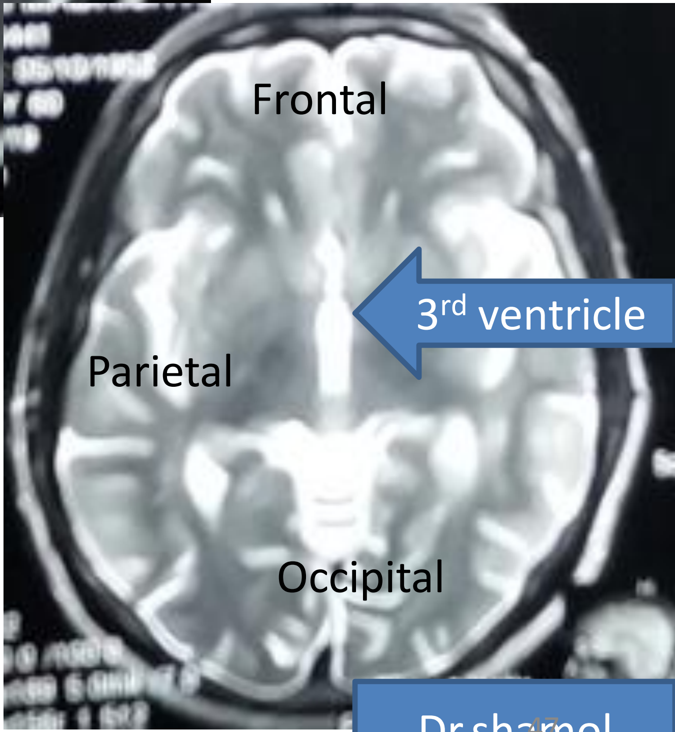

Frontal sinus
Frontal
Mid Brain
Parietal
Occipital
Sylvain sulcus

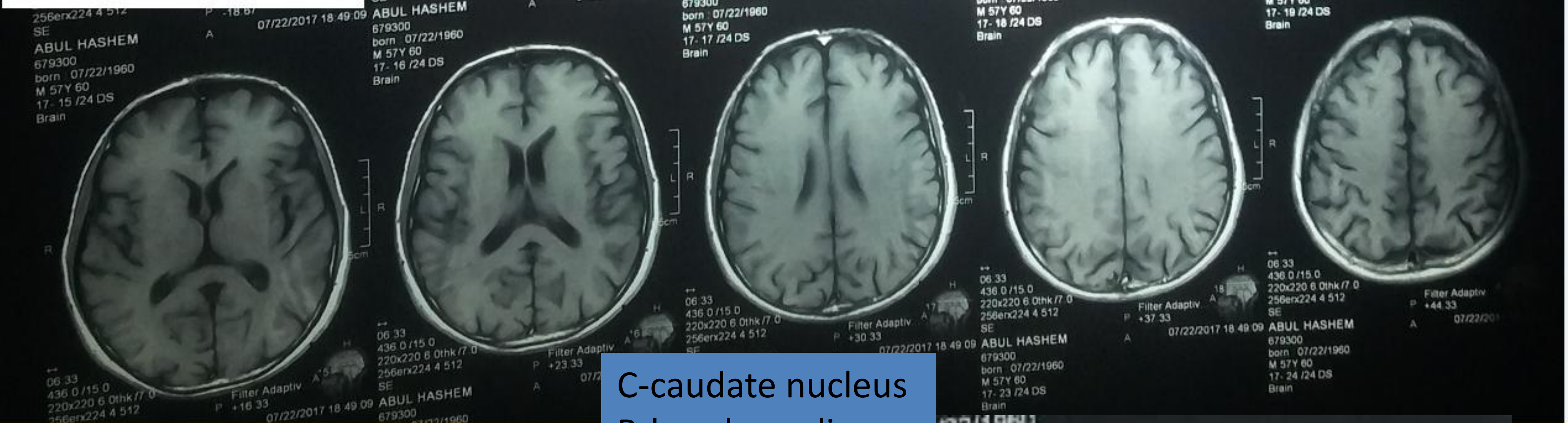

Frontal
Mid Brain
Parietal
Occipital


Frontal
3rd ventricle
Parietal
Occipital
Dr.shamol

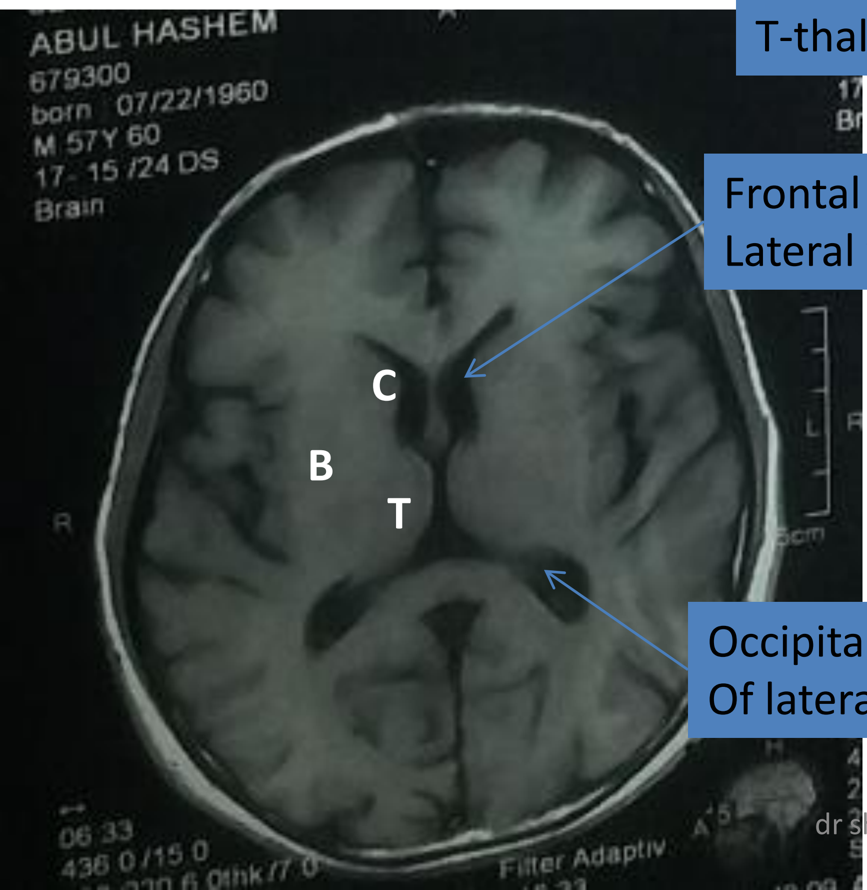

Frontal
Parietal
Mid Brain
Occipital


Frontal
Sylvain sulcus
Parietal
Mid Brain
Occipital


Frontal
3rd ventricle
Parietal
Occipital
Dr.shamol

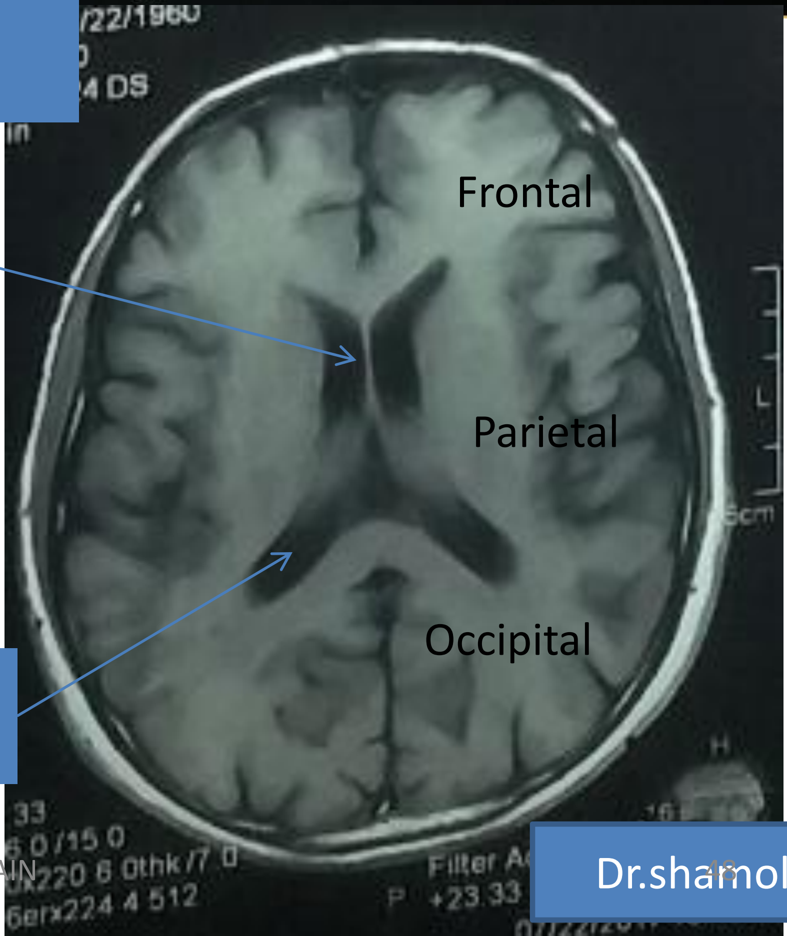


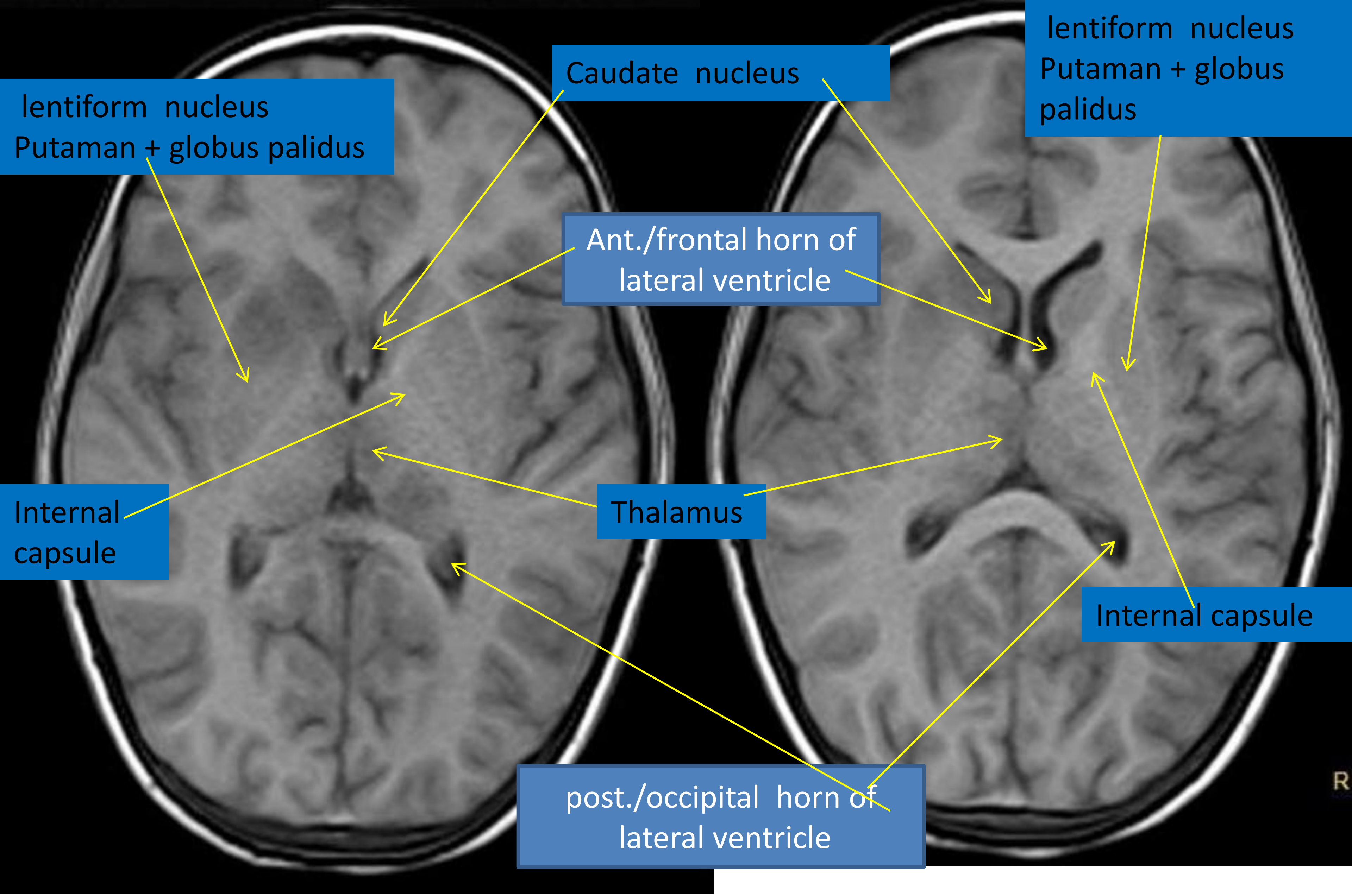
C-caudate nucleus
B-basal ganglia
T-thalamus

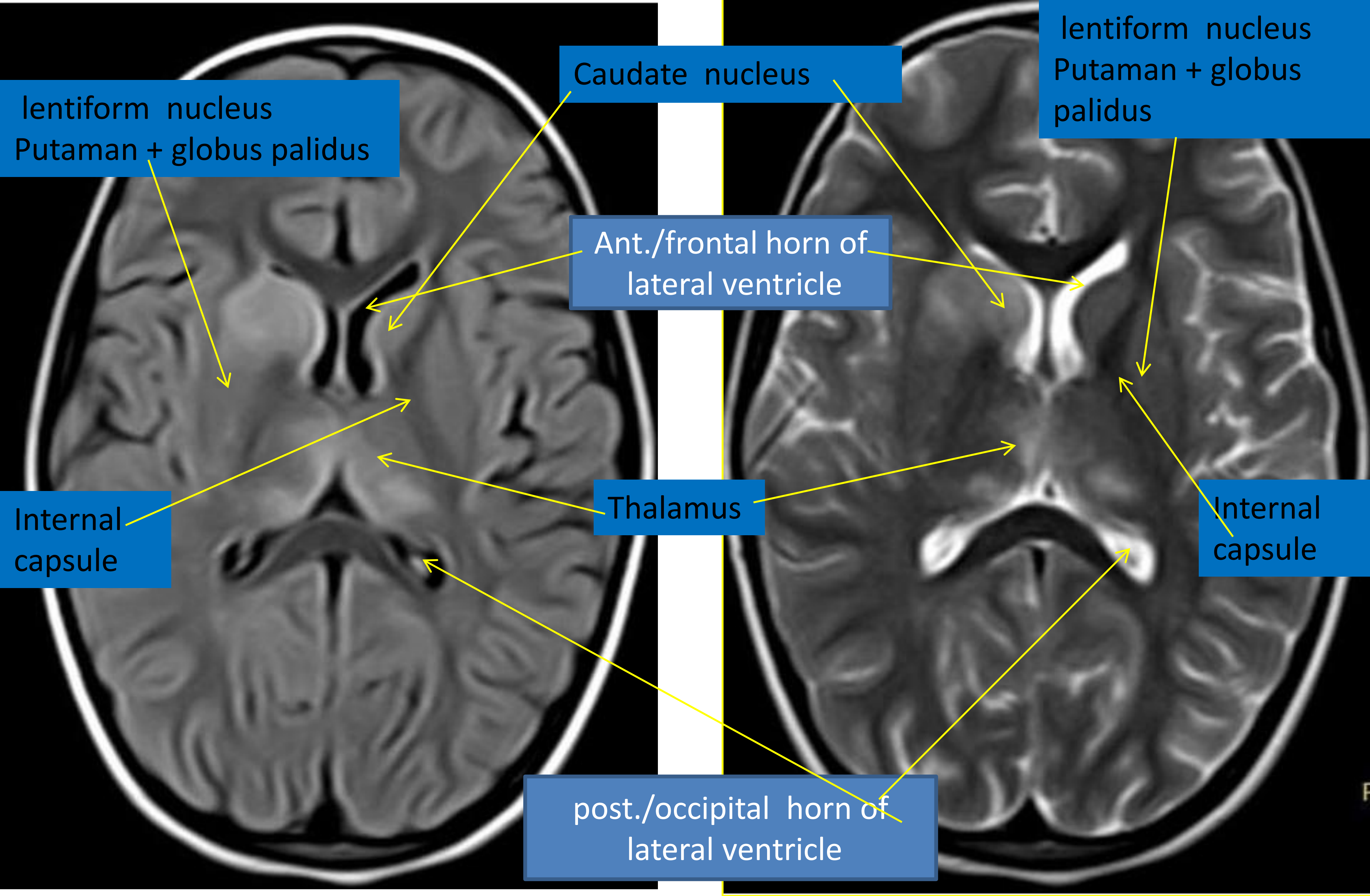


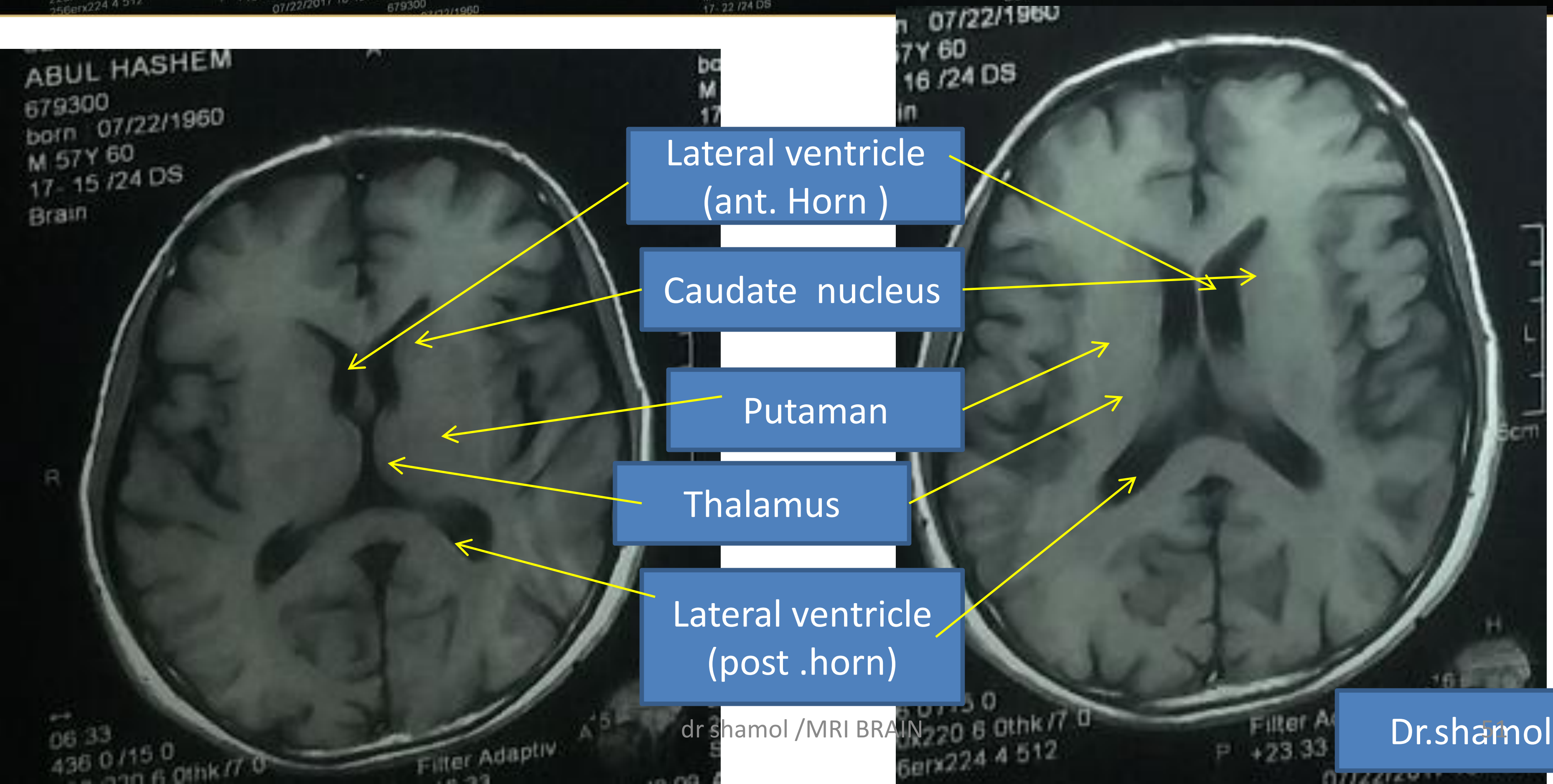
Frontal Horne of Lateral ventricle

Occipital horne Of lateral venticl e

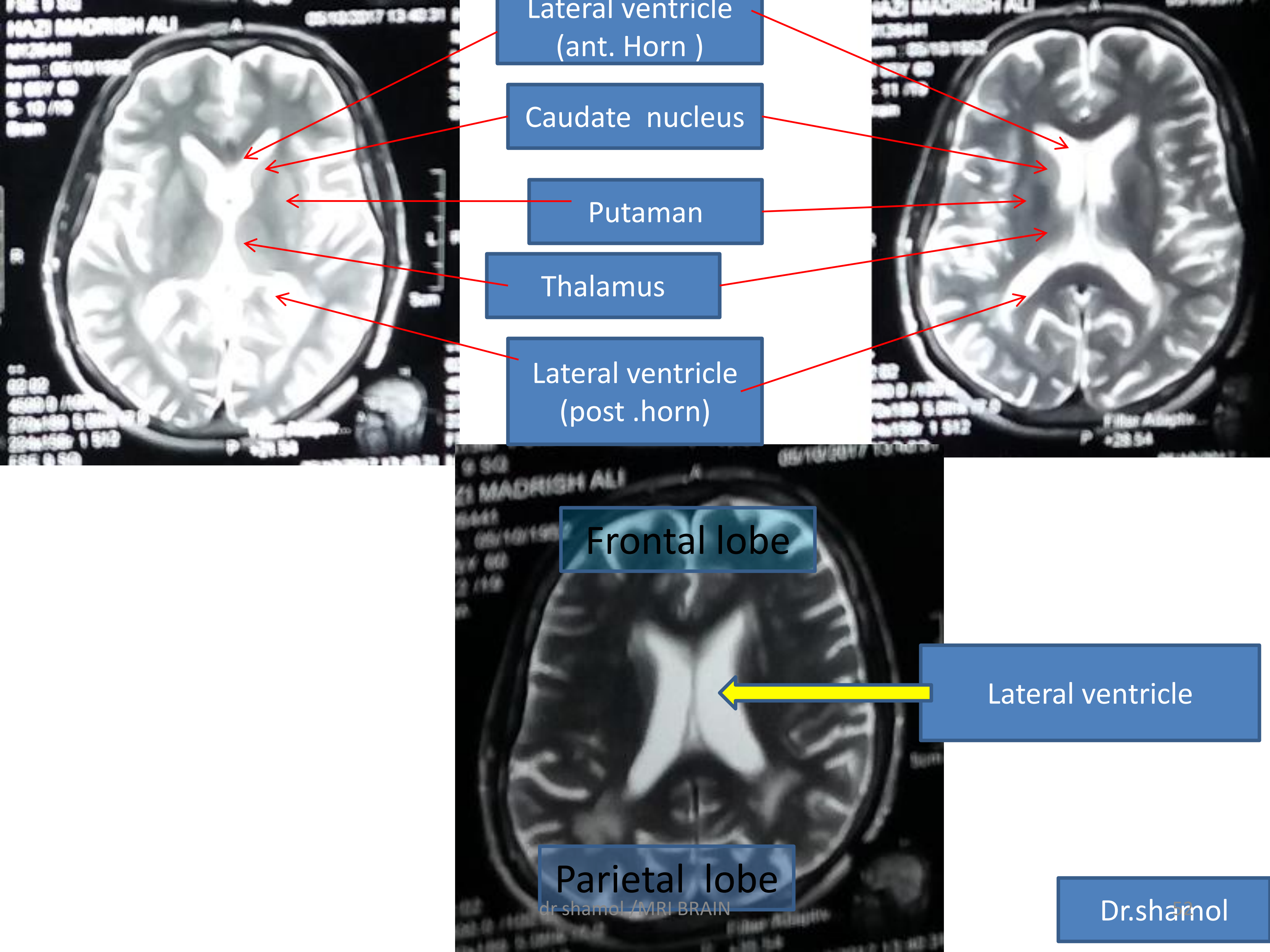


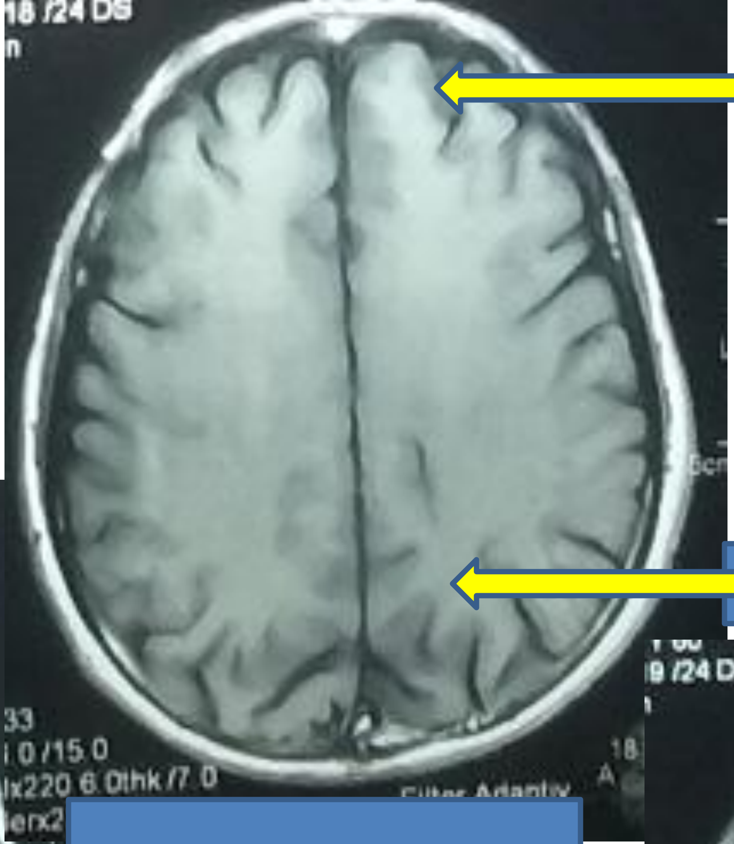

lentiform nucleus
Putaman + globus palidus
Caudate nucleus
lentiform nucleus
Putaman + globus
palidus
Ant./frontal horn of
lateral ventricle
Internal
capsule
Thalamus
Internal capsule
post./occipital horn of
lateral ventricle
R

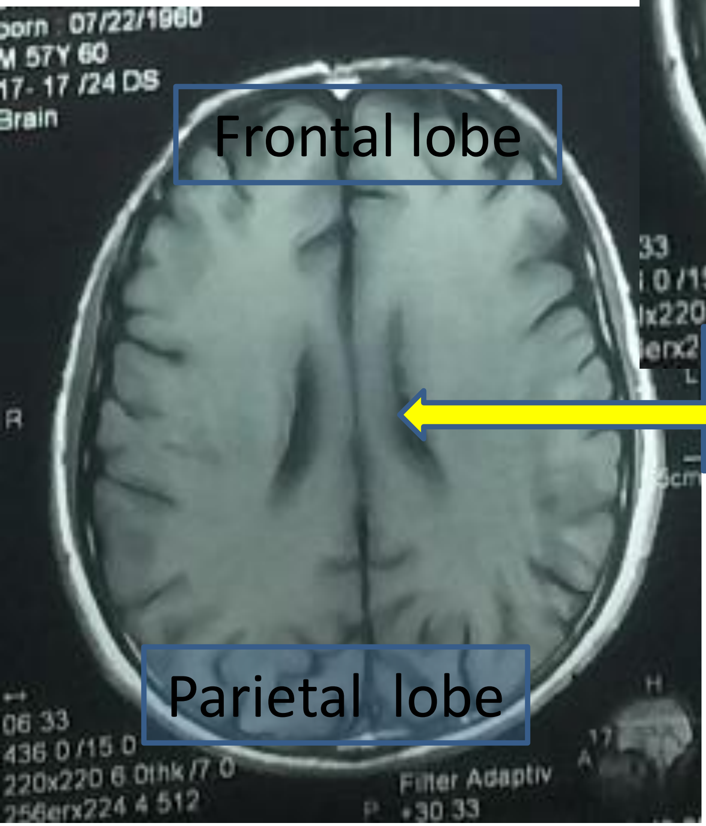

lentiform nucleus
Putaman + globus palidus
Caudate nucleus
lentiform nucleus
Putaman + globus
palidus
Ant./frontal horn of
lateral ventricle
Internal
capsule
Thalamus
Internal
capsule
post./occipital horn of
lateral ventricle

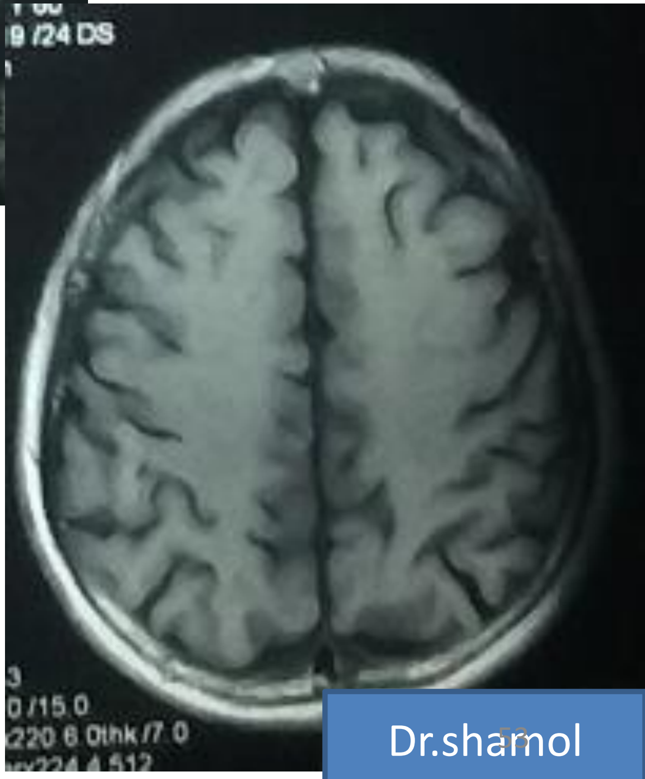

ABUL HASHEM
679300
born 07/22/1960
M 57Y 60
17- 15 /24 DS
Brain
Lateral ventricle (ant. Horn)
Caudate nucleus
Putaman
Thalamus
Lateral ventricle (post .horn)

Dr.shamol

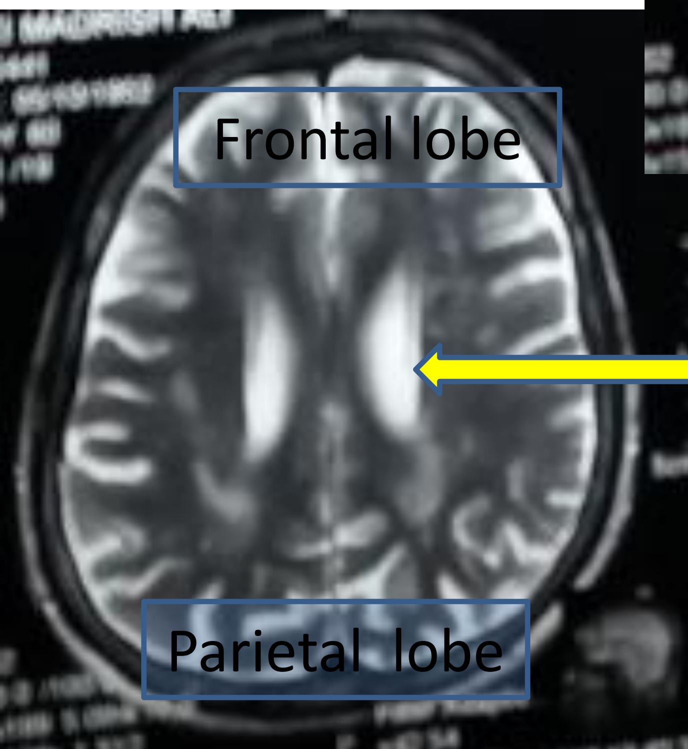

Lateral ventricle (ant. Horn)
Caudate nucleus
Putaman
Thalamus
Lateral ventricle (post .horn)
Frontal lobe
Lateral ventricle
Parietal lobe
Dr.shamol


Frontal lobe
Parietal lobe
18 /24 DS
33
Filter Adaptiv

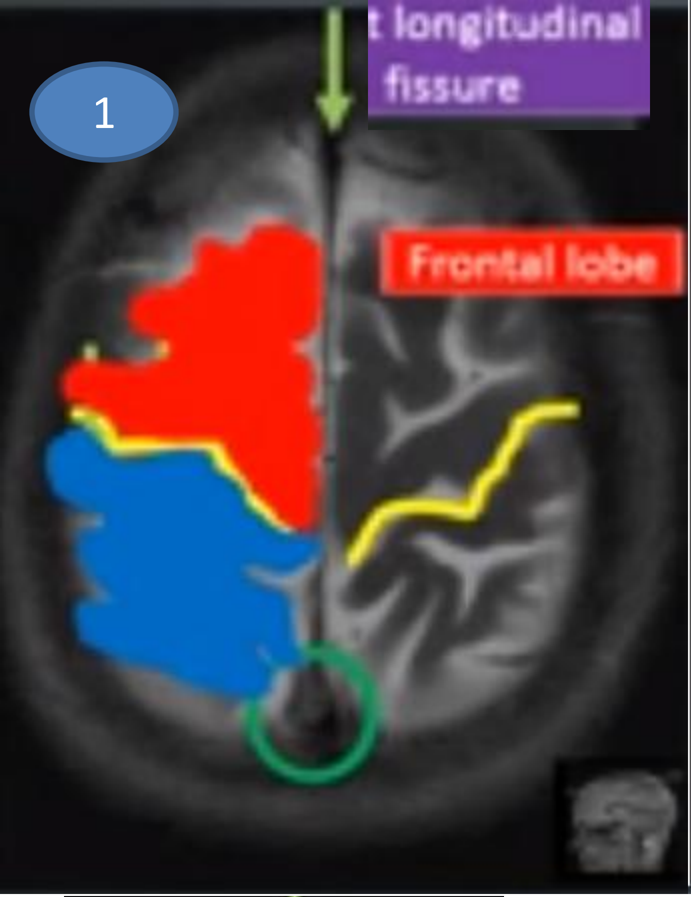

born 07/22/1960
M 57Y 60
17- 17 /24 DS
Brain
Frontal lobe
Lateral ventricle
R
Parietal lobe
06 33
436 0 /15 0
220x220 6 0thk /7 0
256erx224 4 512
Filter Adaptiv
P +30 33

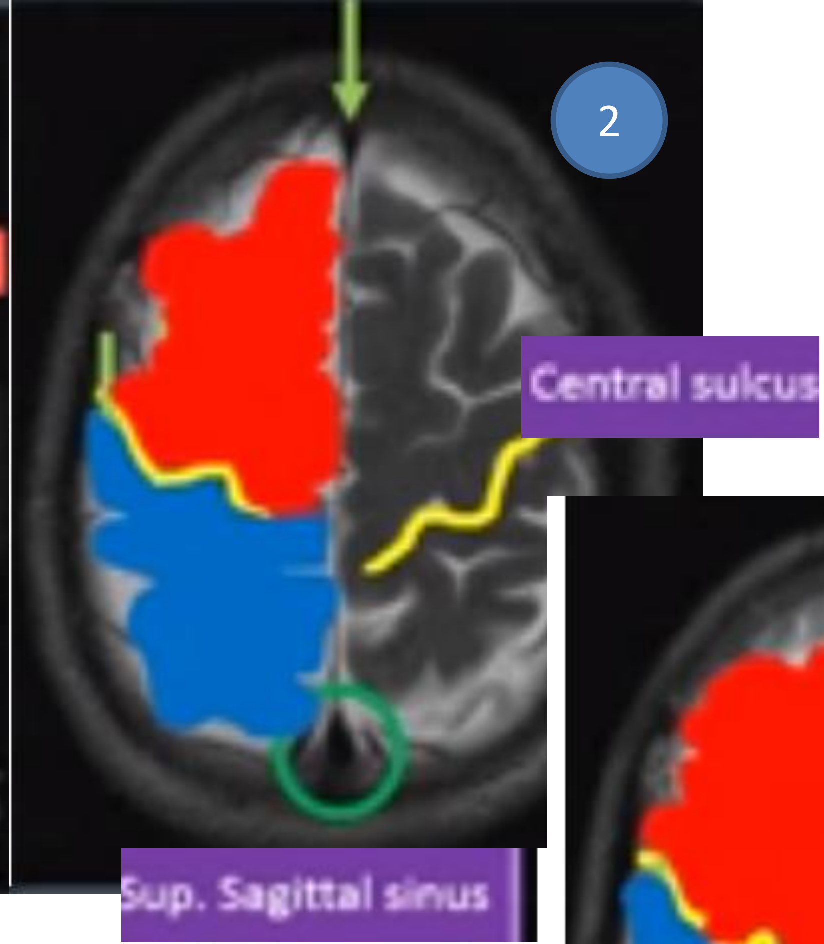

9 /24 DS
0 /15 0
220 6 0thk /7 0
Dr.shamol

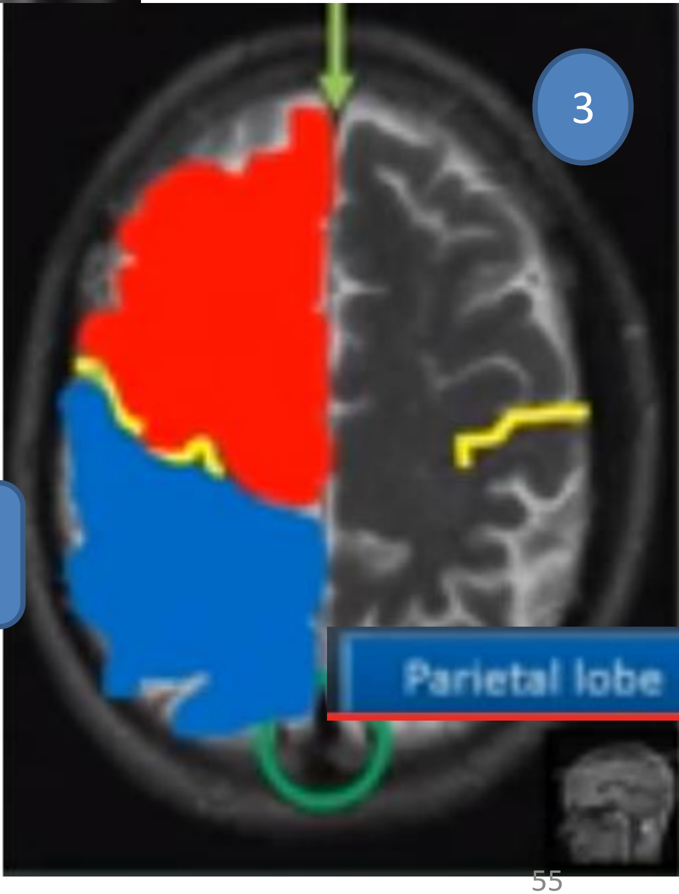

Frontal lobe
Parietal lobe

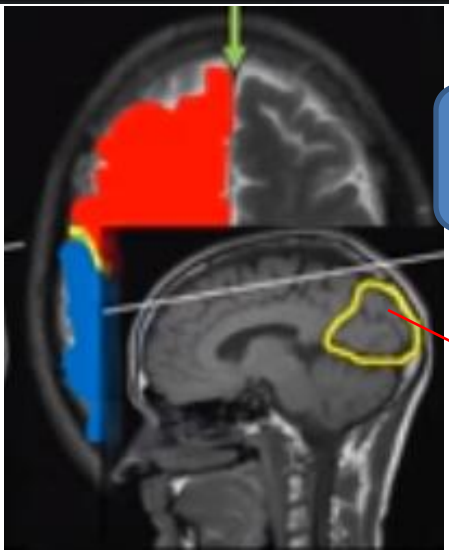

Frontal lobe
Parietal lobe
Lateral ventricle


Dr.shamol



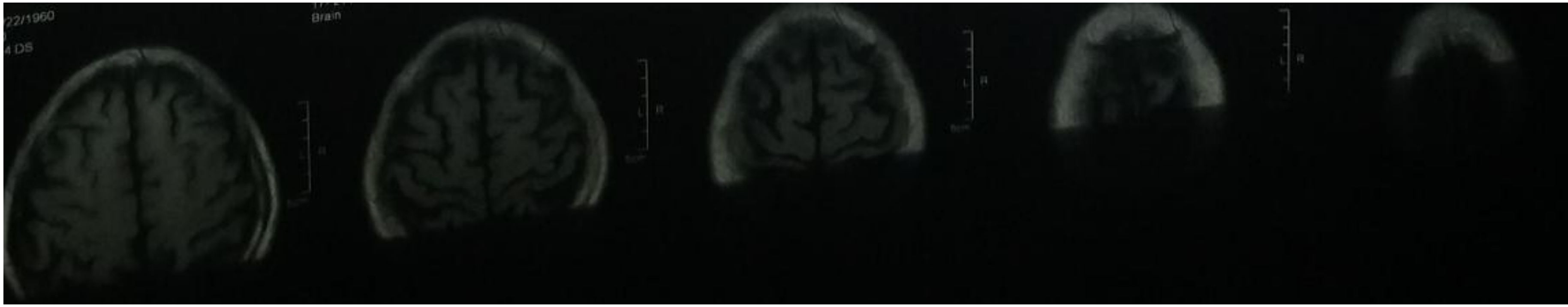


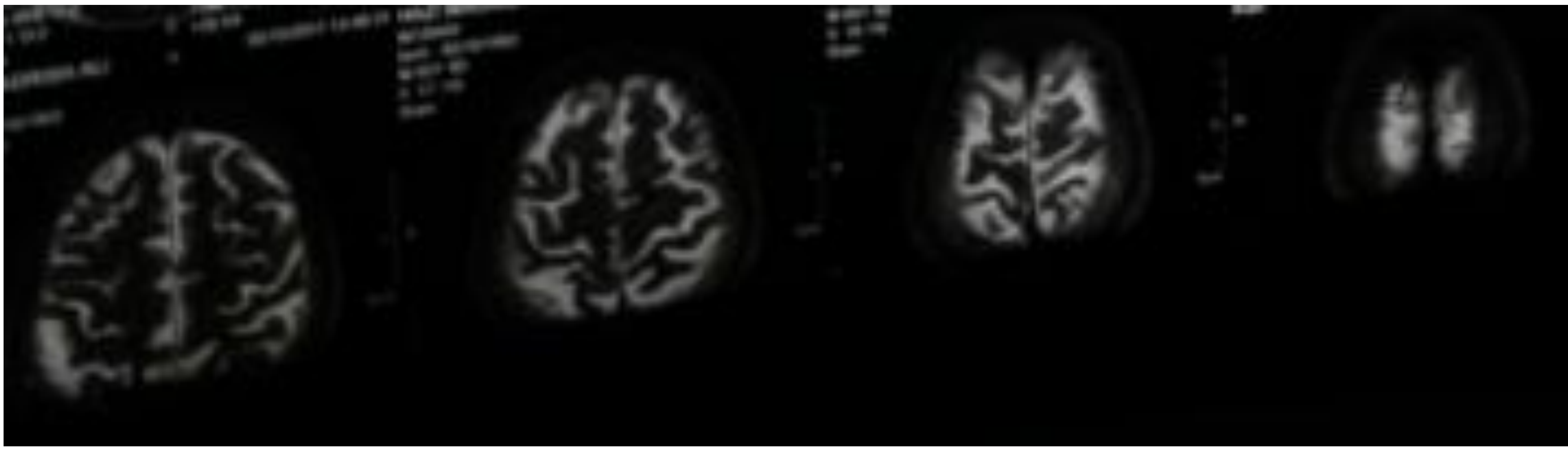




Here no occipital lobe
It remain below the section line

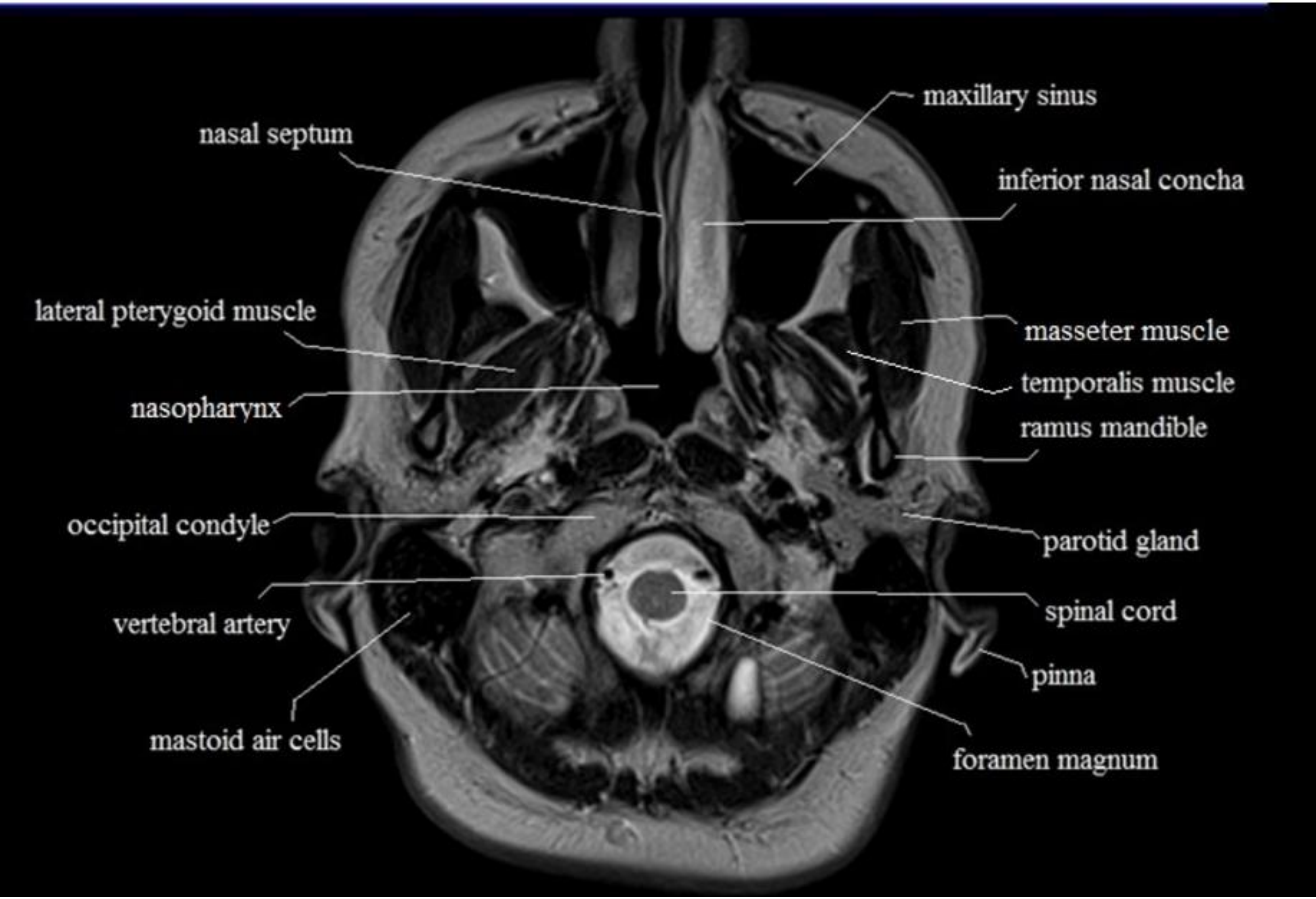
Occipital lobe

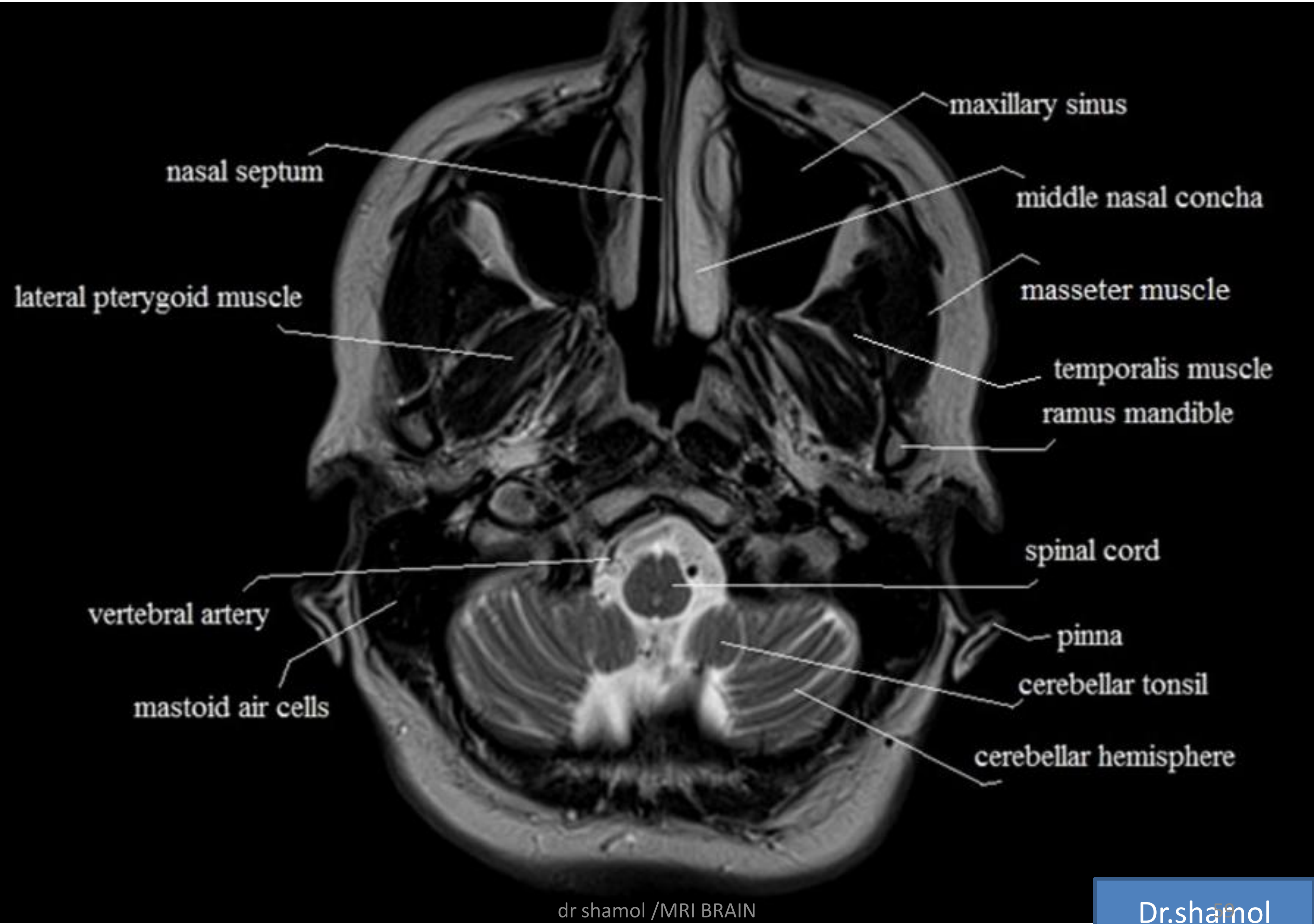

22/1960
4 DS
Brain

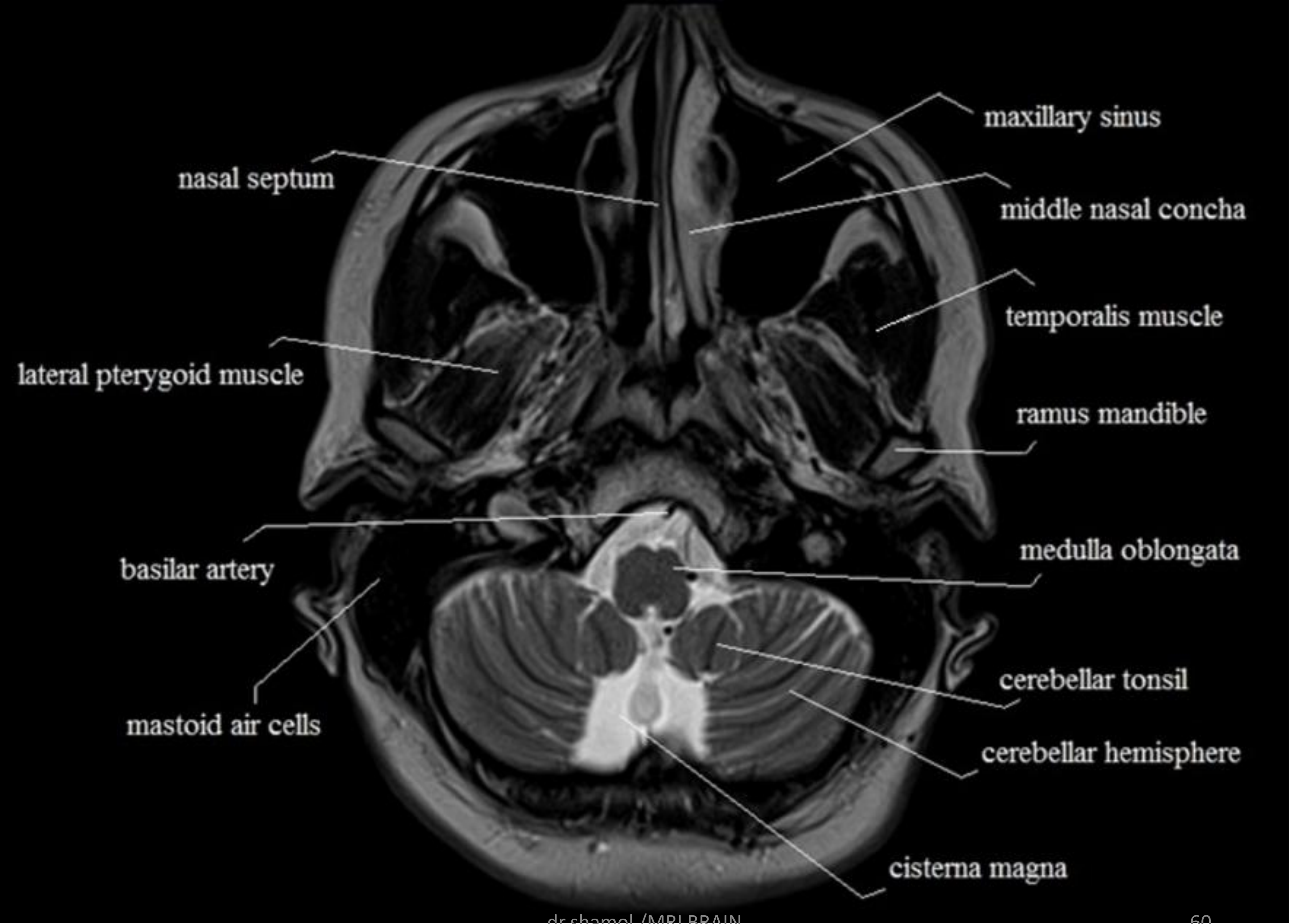


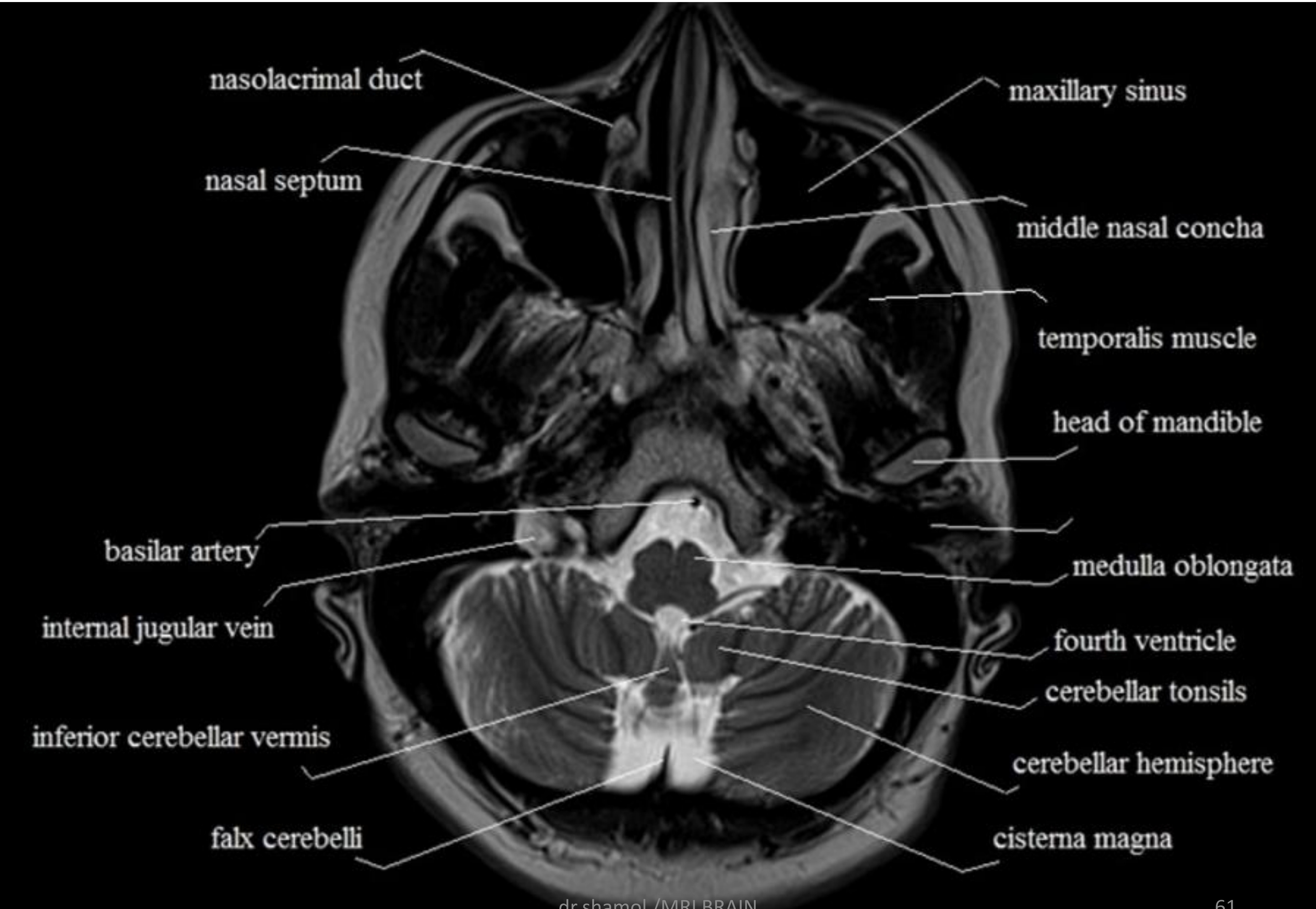
AXIAL
Structure A to Z

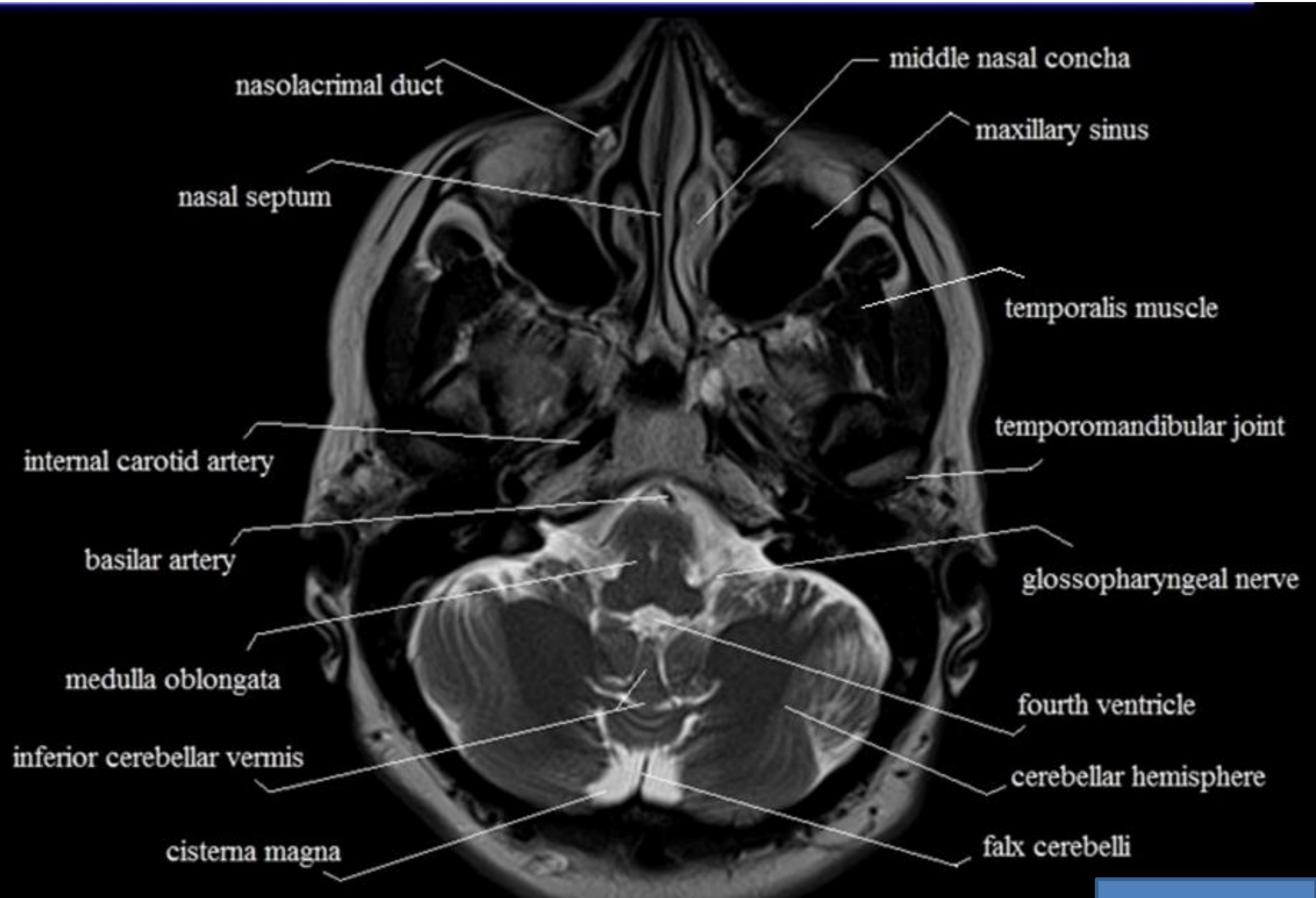
Sequence
FROM BELLOW → UPWARD

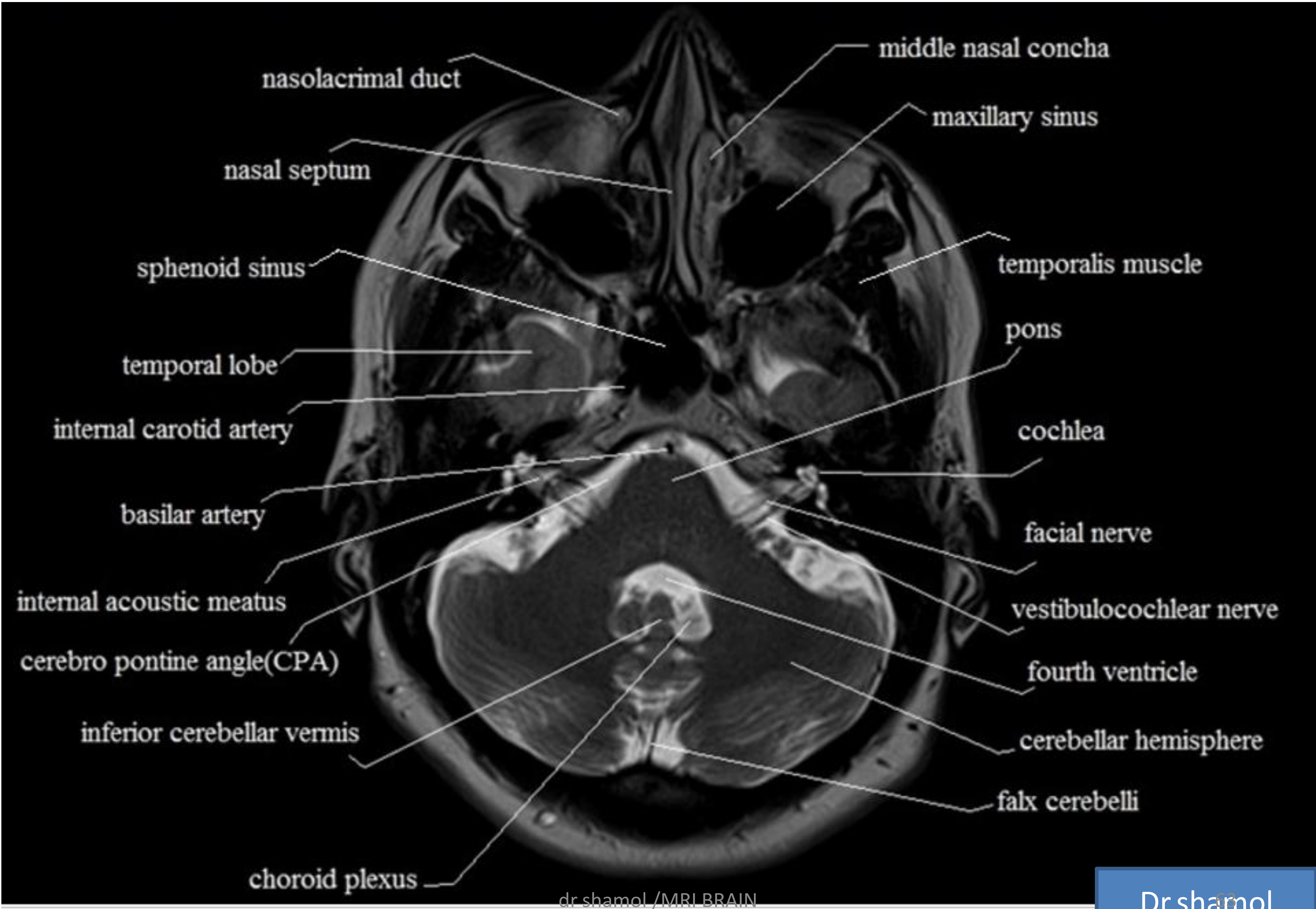

maxillary sinus
nasal septum
inferior nasal concha
lateral pterygoid muscle
masseter muscle
temporalis muscle
nasopharynx
ramus mandible
occipital condyle
parotid gland
vertebral artery
spinal cord
pinna
mastoid air cells
foramen magnum

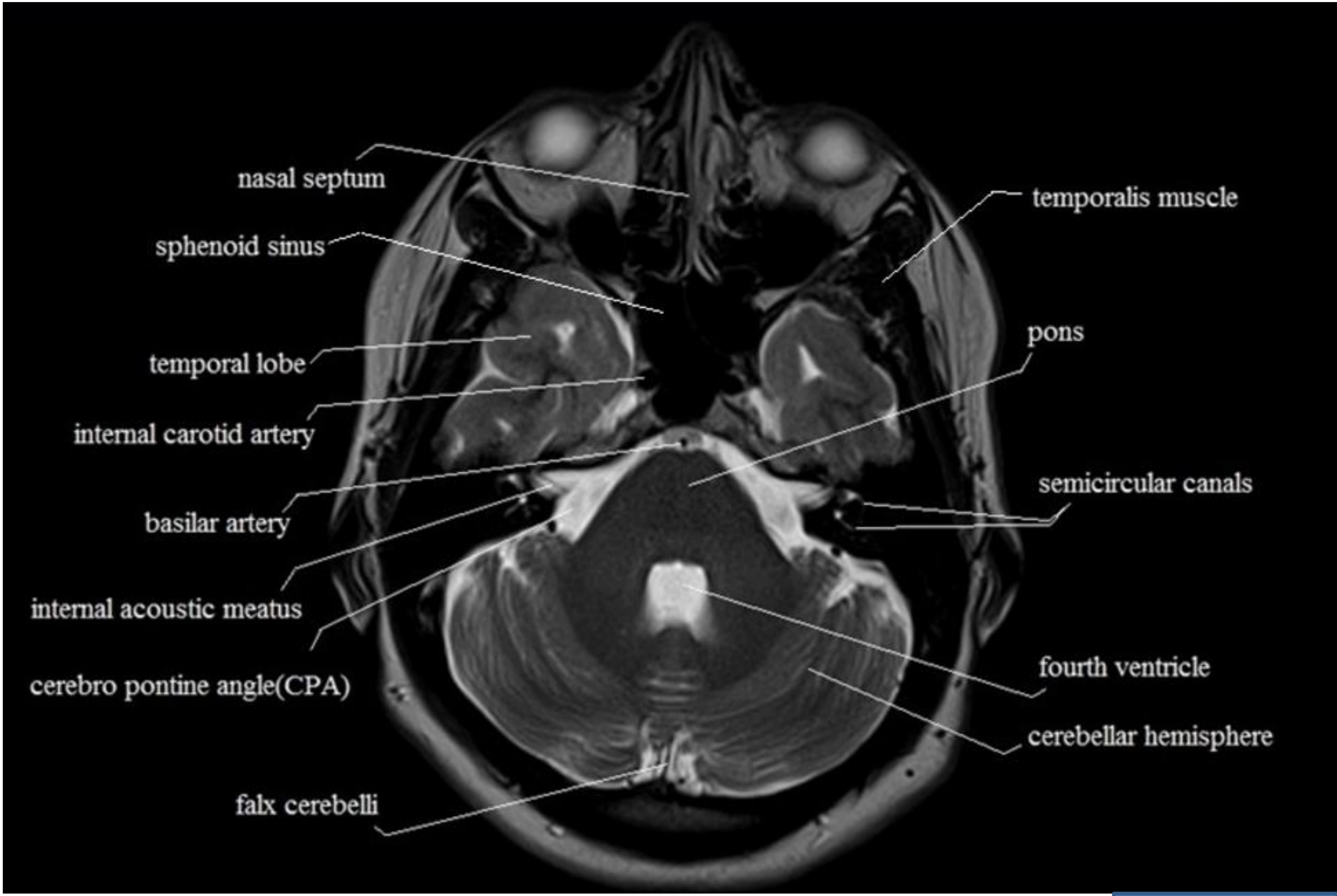

maxillary sinus
nasal septum
middle nasal concha
lateral pterygoid muscle
masseter muscle
temporalis muscle
ramus mandible
spinal cord
vertebral artery
pinna
mastoid air cells
cerebellar tonsil
cerebellar hemisphere

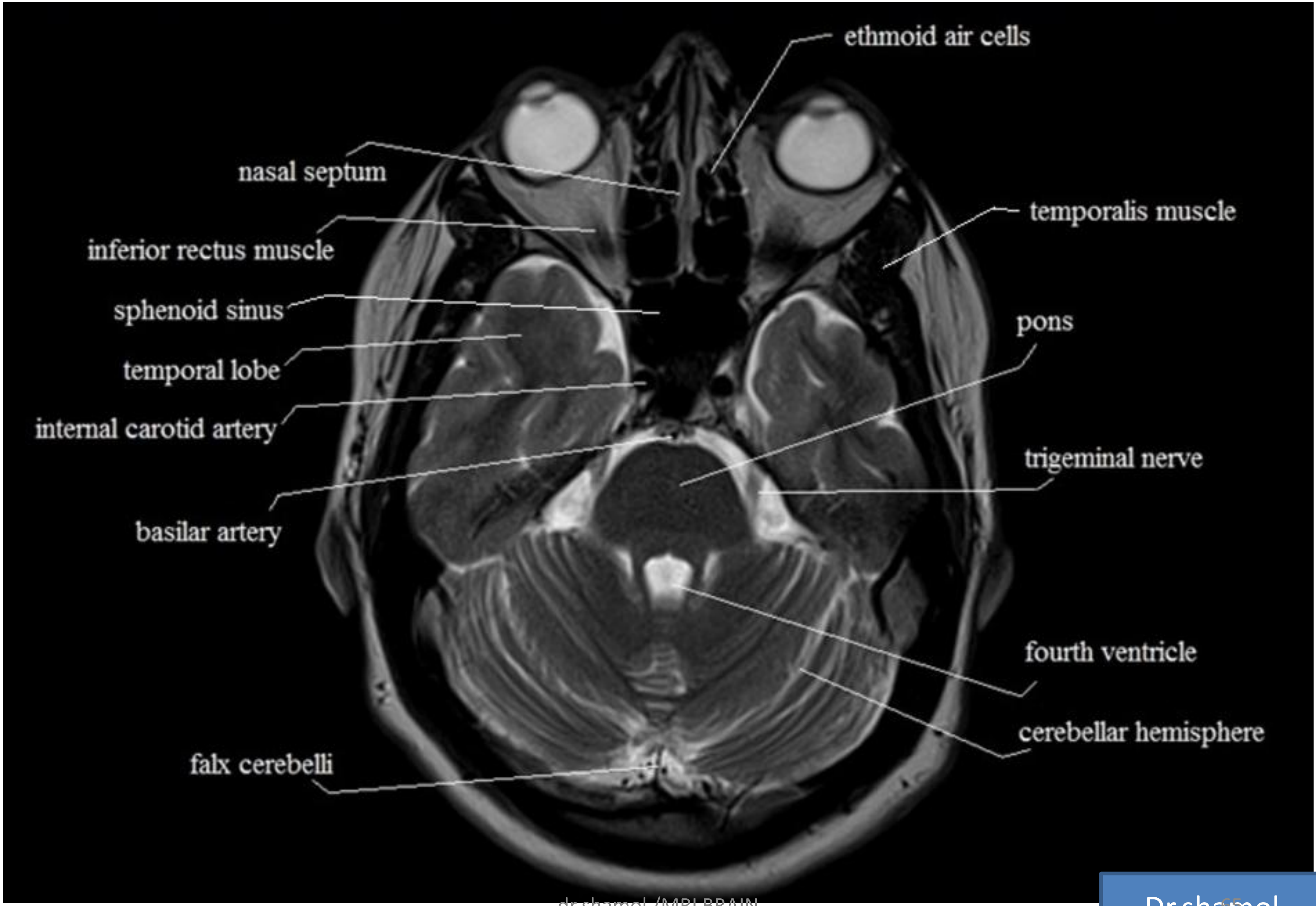

maxillary sinus
nasal septum
middle nasal concha
temporalis muscle
lateral pterygoid muscle
ramus mandible
basilar artery
medulla oblongata
mastoid air cells
cerebellar tonsil
cerebellar hemisphere
cisterna magna


nasolacrimal duct
maxillary sinus
nasal septum
middle nasal concha
temporalis muscle
head of mandible
basilar artery
medulla oblongata
internal jugular vein
fourth ventricle
cerebellar tonsils
inferior cerebellar vermis
cerebellar hemisphere
falx cerebelli
cisterna magna

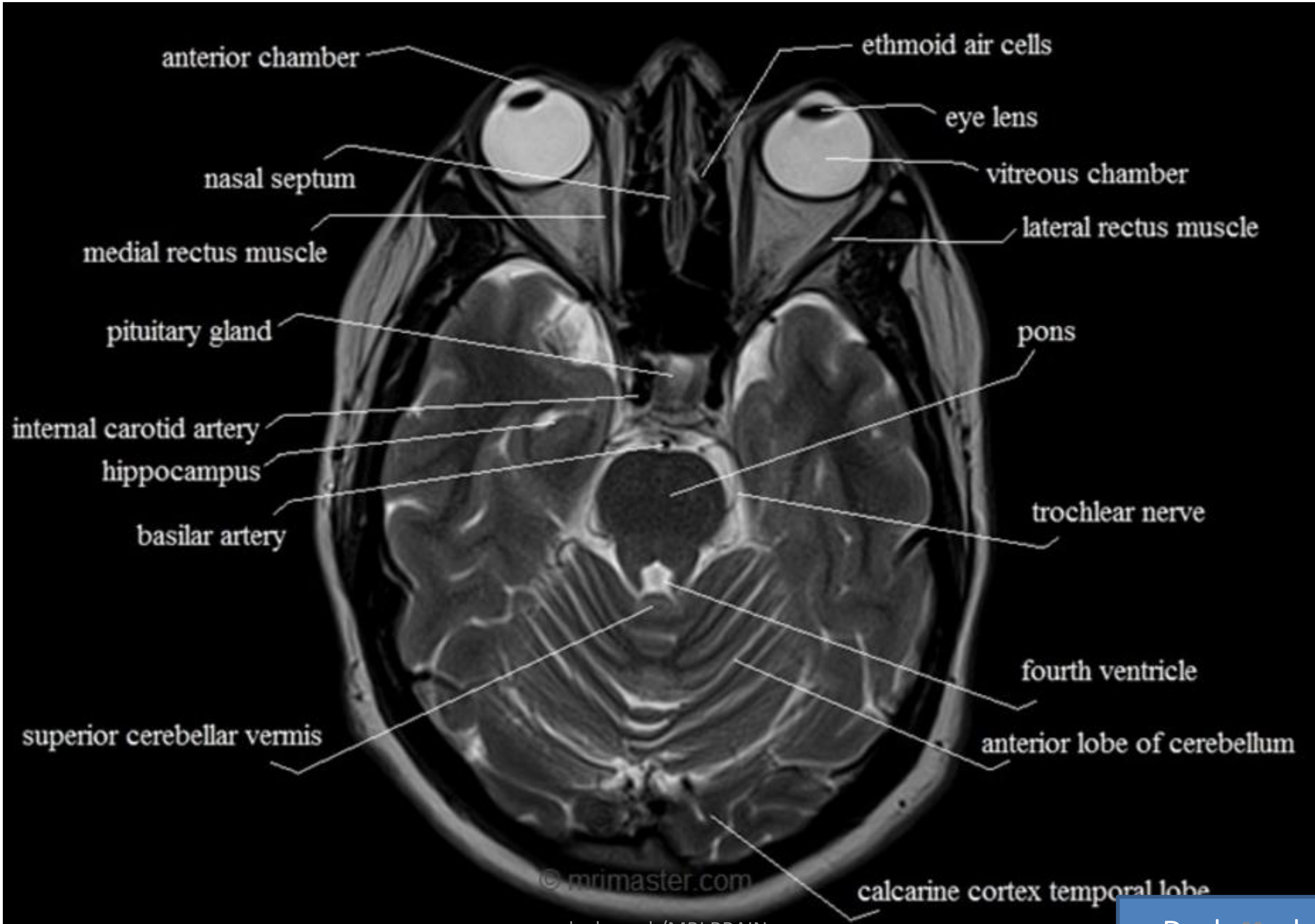

nasolacrimal duct
middle nasal concha
maxillary sinus
nasal septum
temporalis muscle
temporomandibular joint
internal carotid artery
basilar artery
glossopharyngeal nerve
medulla oblongata
fourth ventricle
inferior cerebellar vermis
cerebellar hemisphere
cisterna magna
falx cerebelli


middle nasal concha
nasolacrimal duct
maxillary sinus
nasal septum
sphenoid sinus
temporalis muscle
pons
temporal lobe
internal carotid artery
cochlea
basilar artery
facial nerve
internal acoustic meatus
vestibulocochlear nerve
cerebro pontine angle(CPA)
fourth ventricle
inferior cerebellar vermis
cerebellar hemisphere
falx cerebelli
choroid plexus


nasal septum
sphenoid sinus
temporal lobe
internal carotid artery
basilar artery
internal acoustic meatus
cerebro pontine angle(CPA)
falx cerebelli
temporalis muscle
pons
semicircular canals
fourth ventricle
cerebellar hemisphere


ethmoid air cells
nasal septum
temporalis muscle
inferior rectus muscle
sphenoid sinus
pons
temporal lobe
internal carotid artery
trigeminal nerve
basilar artery
fourth ventricle
cerebellar hemisphere
falx cerebelli

Dr.shamol


anterior chamber
ethmoid air cells
eye lens
nasal septum
vitreous chamber
lateral rectus muscle
medial rectus muscle
pituitary gland
pons
internal carotid artery
hippocampus
basilar artery
trochlear nerve
fourth ventricle
superior cerebellar vermis
anterior lobe of cerebellum
calcarine cortex temporal lobe
© mrimaster.com

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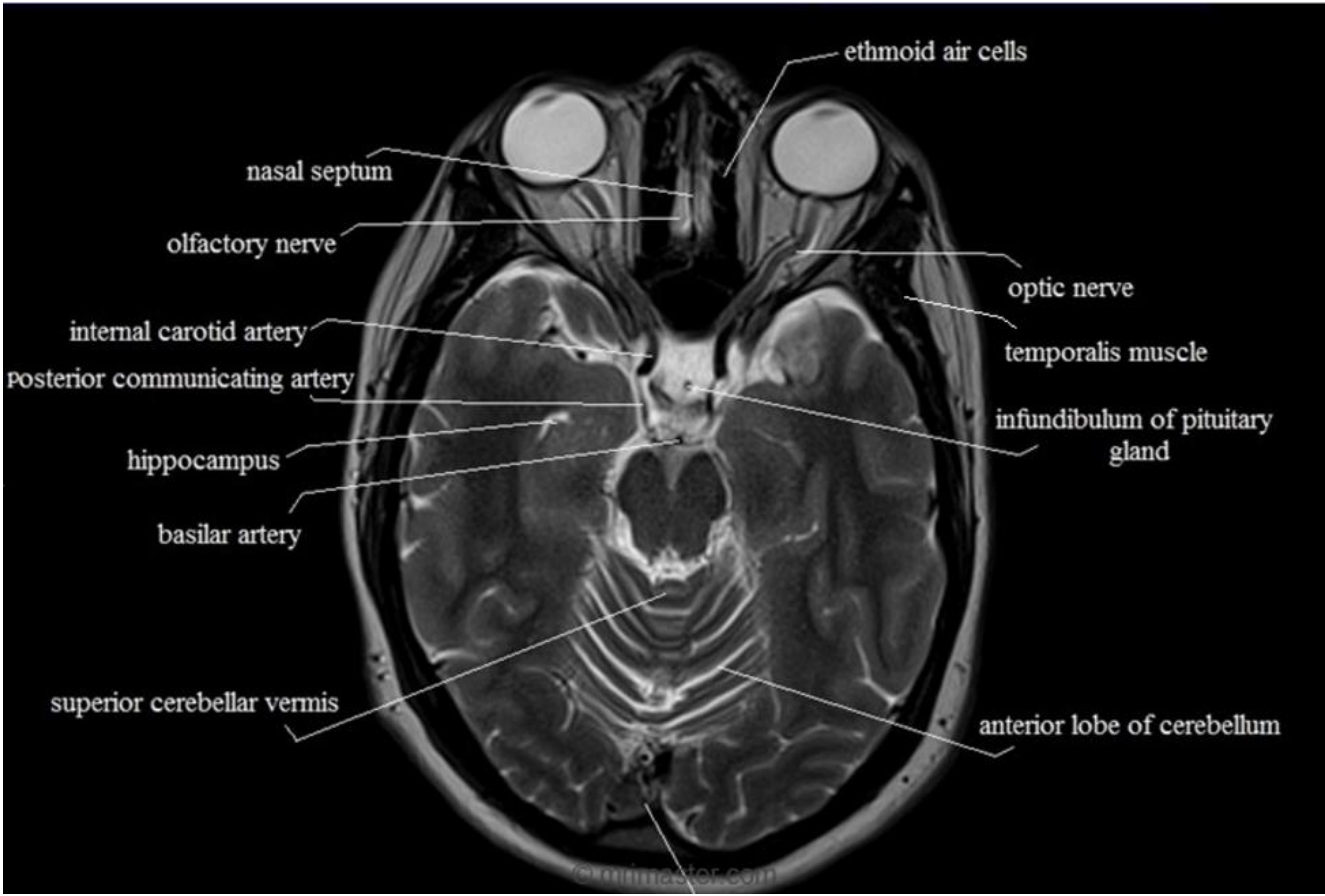
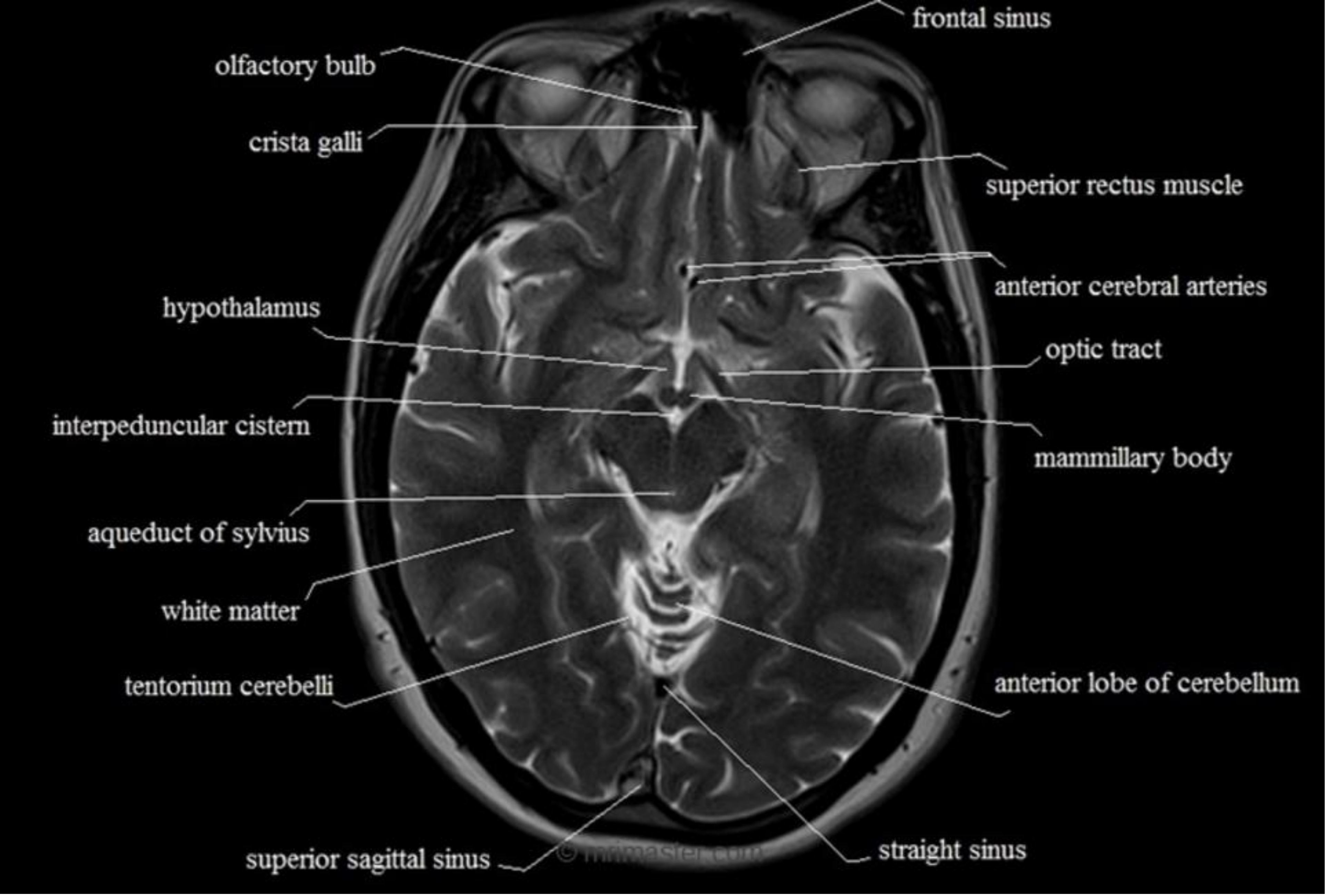
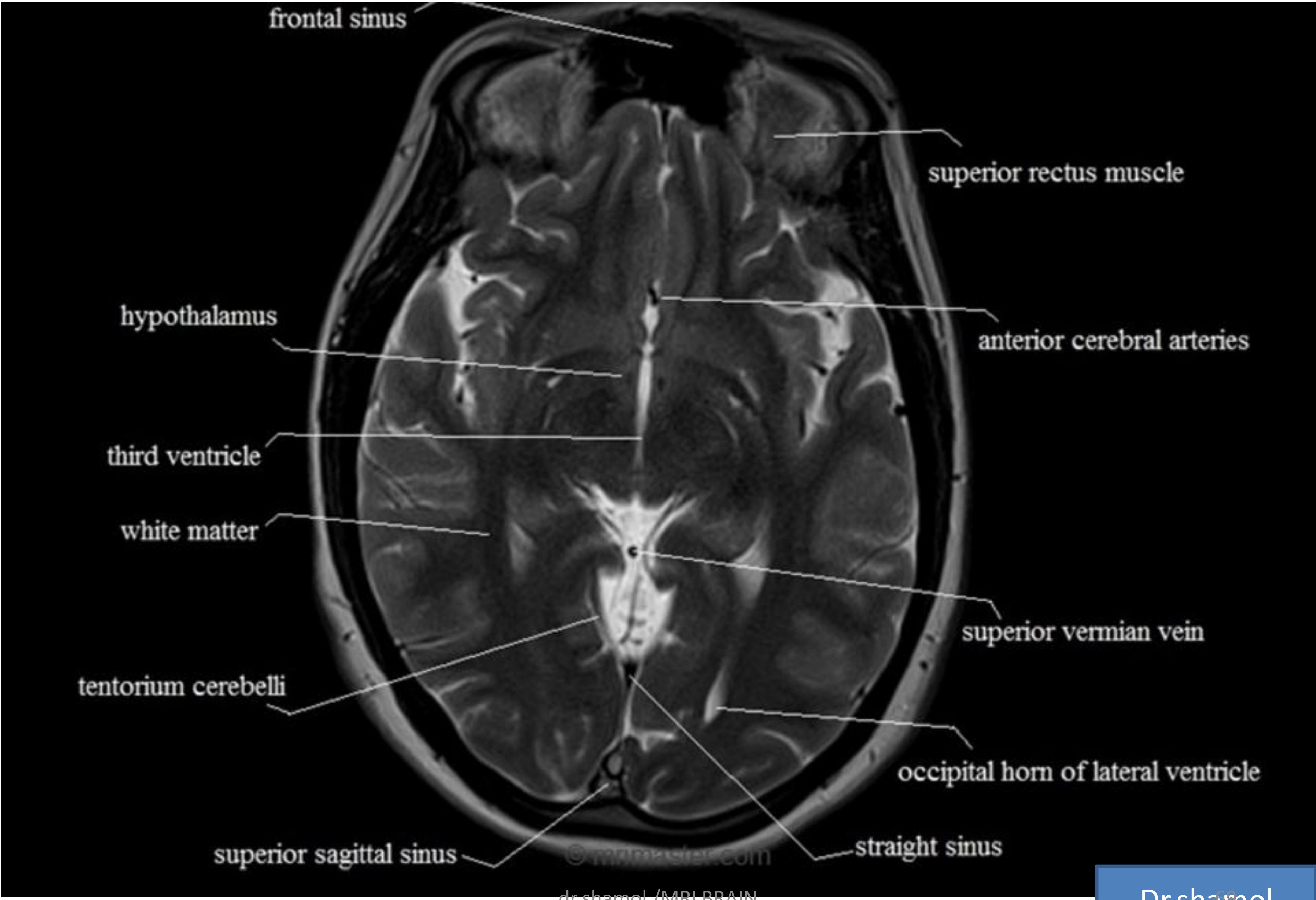
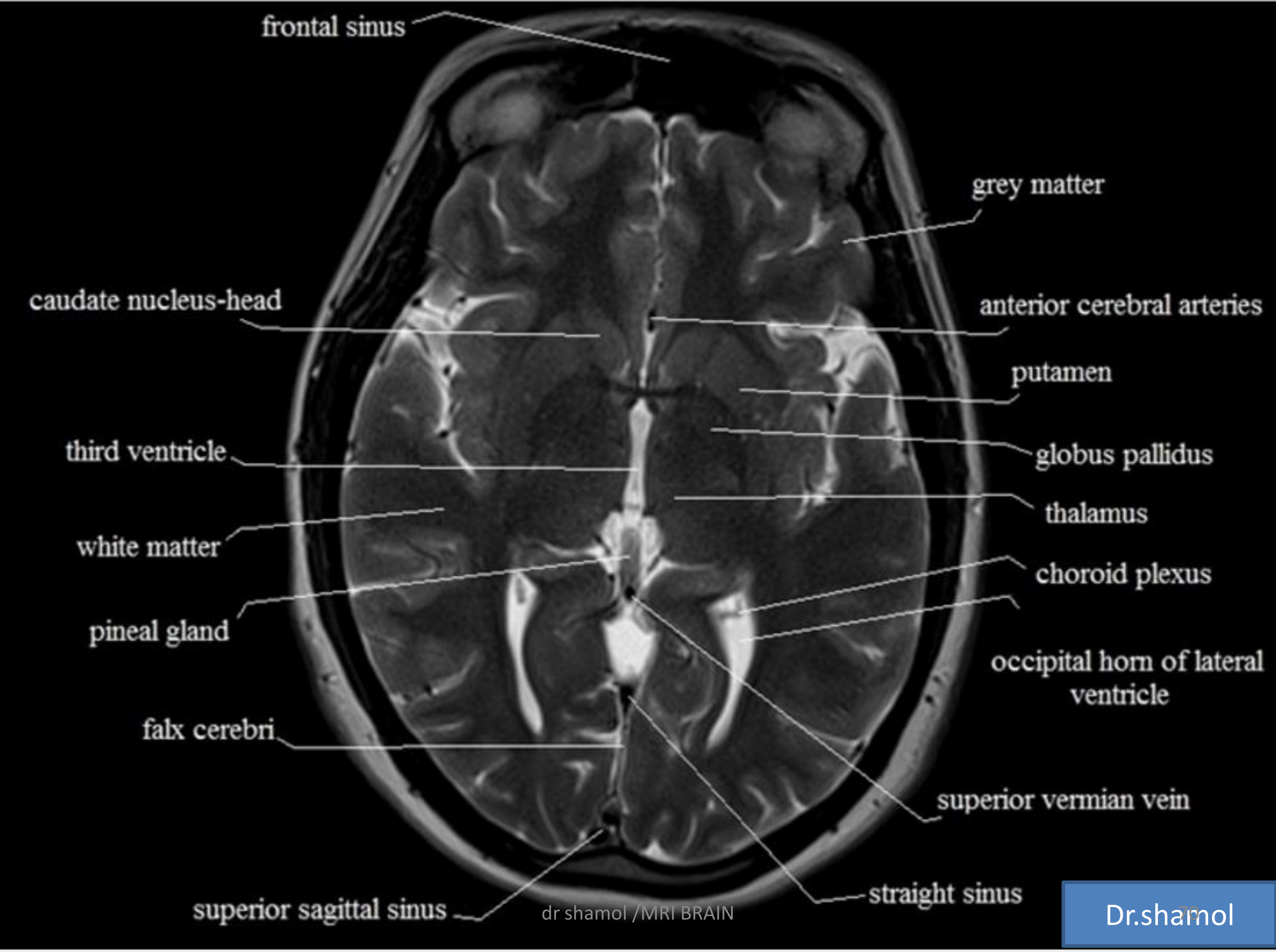

ethmoid air cells
nasal septum
olfactory nerve
optic nerve
temporalis muscle
internal carotid artery
Posterior communicating artery
infundibulum of pituitary gland
hippocampus
basilar artery
superior cerebellar vermis
anterior lobe of cerebellum

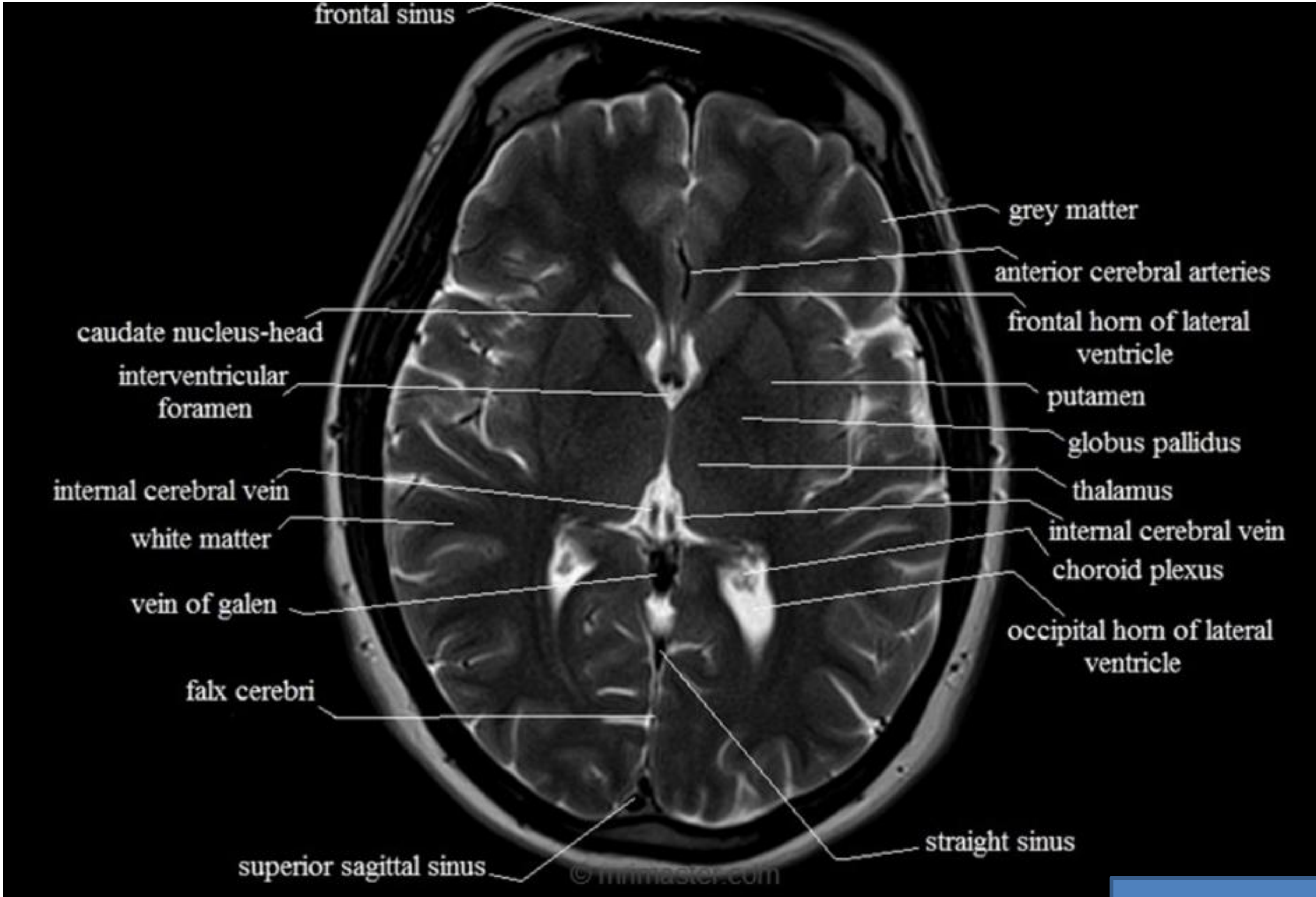

frontal sinus
olfactory bulb
crista galli
superior rectus muscle
anterior cerebral arteries
hypothalamus
optic tract
interpeduncular cistern
mammillary body
aqueduct of sylvius
white matter
tentorium cerebelli
anterior lobe of cerebellum
superior sagittal sinus
straight sinus
© mrimaster.com

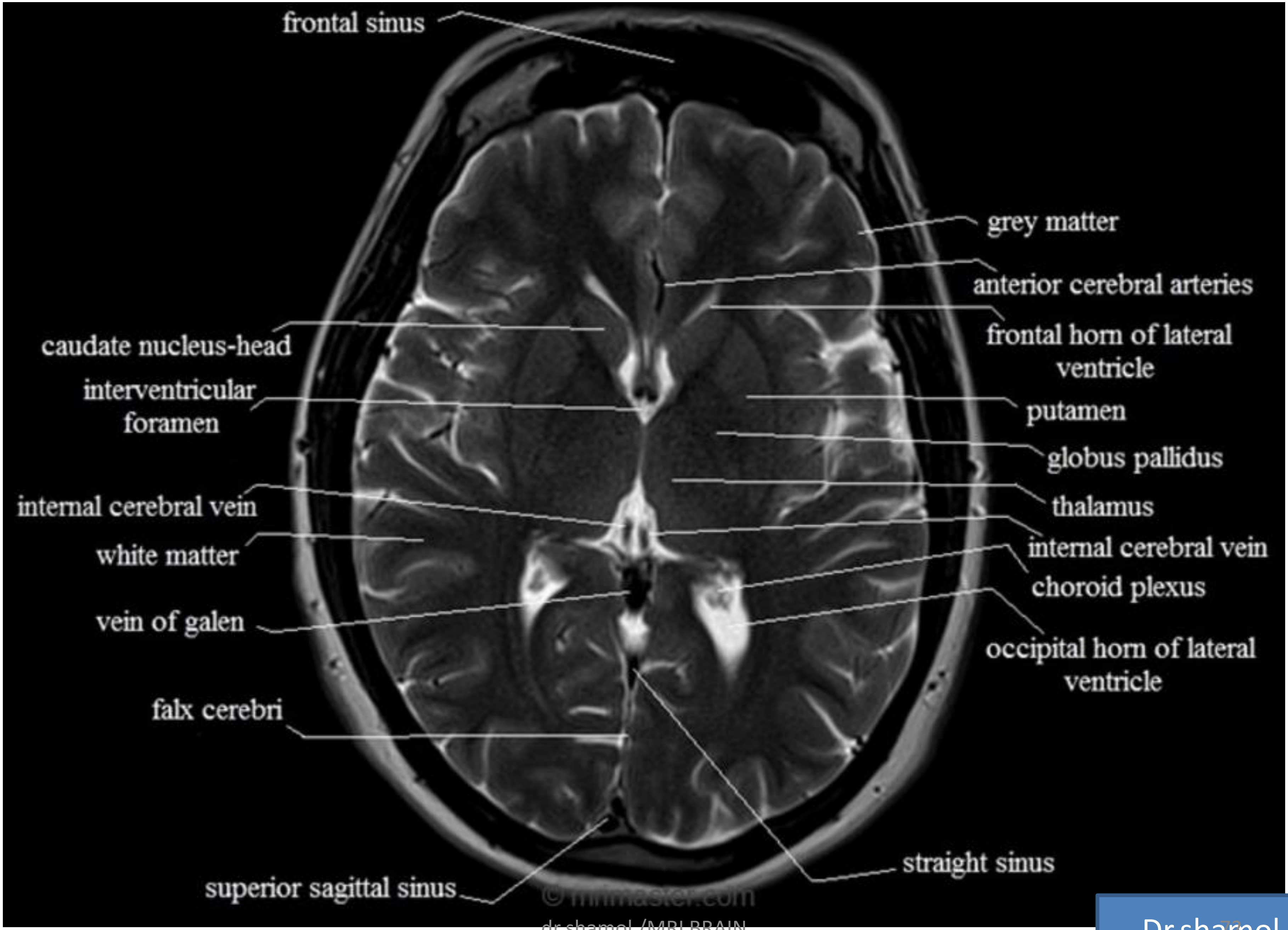


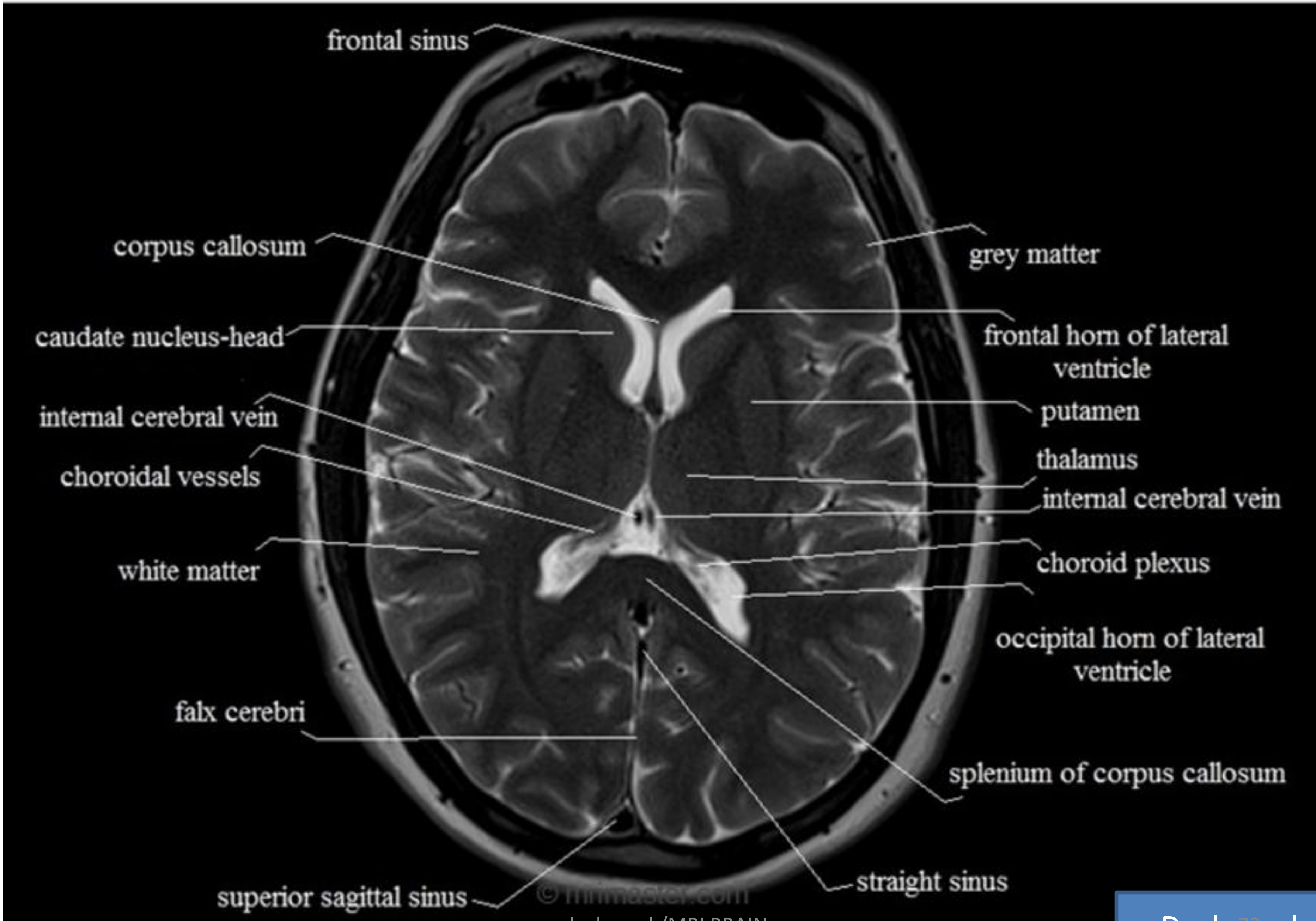
frontal sinus
superior rectus muscle
hypothalamus
anterior cerebral arteries
third ventricle
white matter
superior vermian vein
tentorium cerebelli
occipital horn of lateral ventricle
superior sagittal sinus
straight sinus

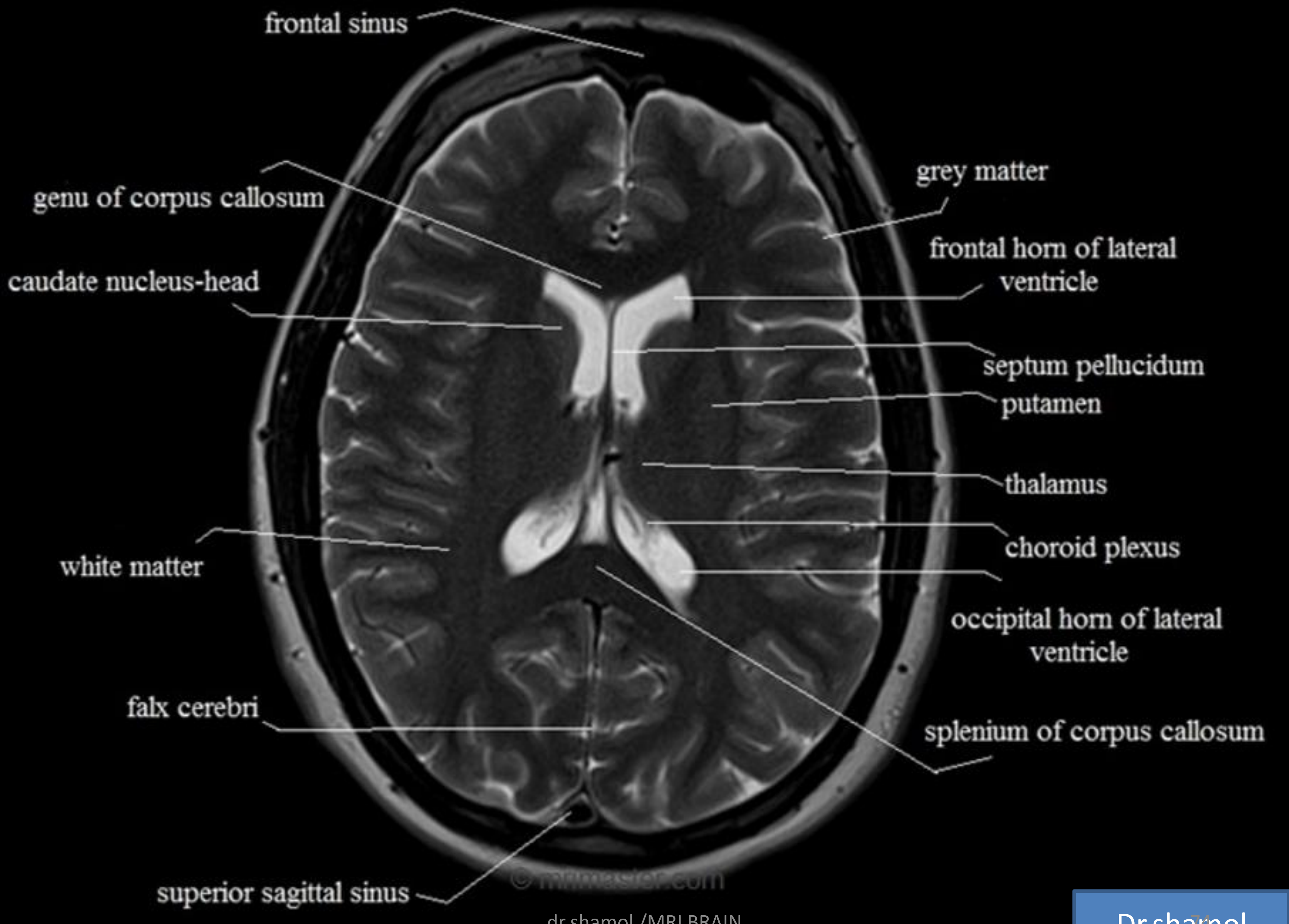

frontal sinus
grey matter
caudate nucleus-head
anterior cerebral arteries
putamen
third ventricle
globus pallidus
thalamus
white matter
choroid plexus
pineal gland
occipital horn of lateral ventricle
falx cerebri
superior vermian vein
straight sinus
superior sagittal sinus

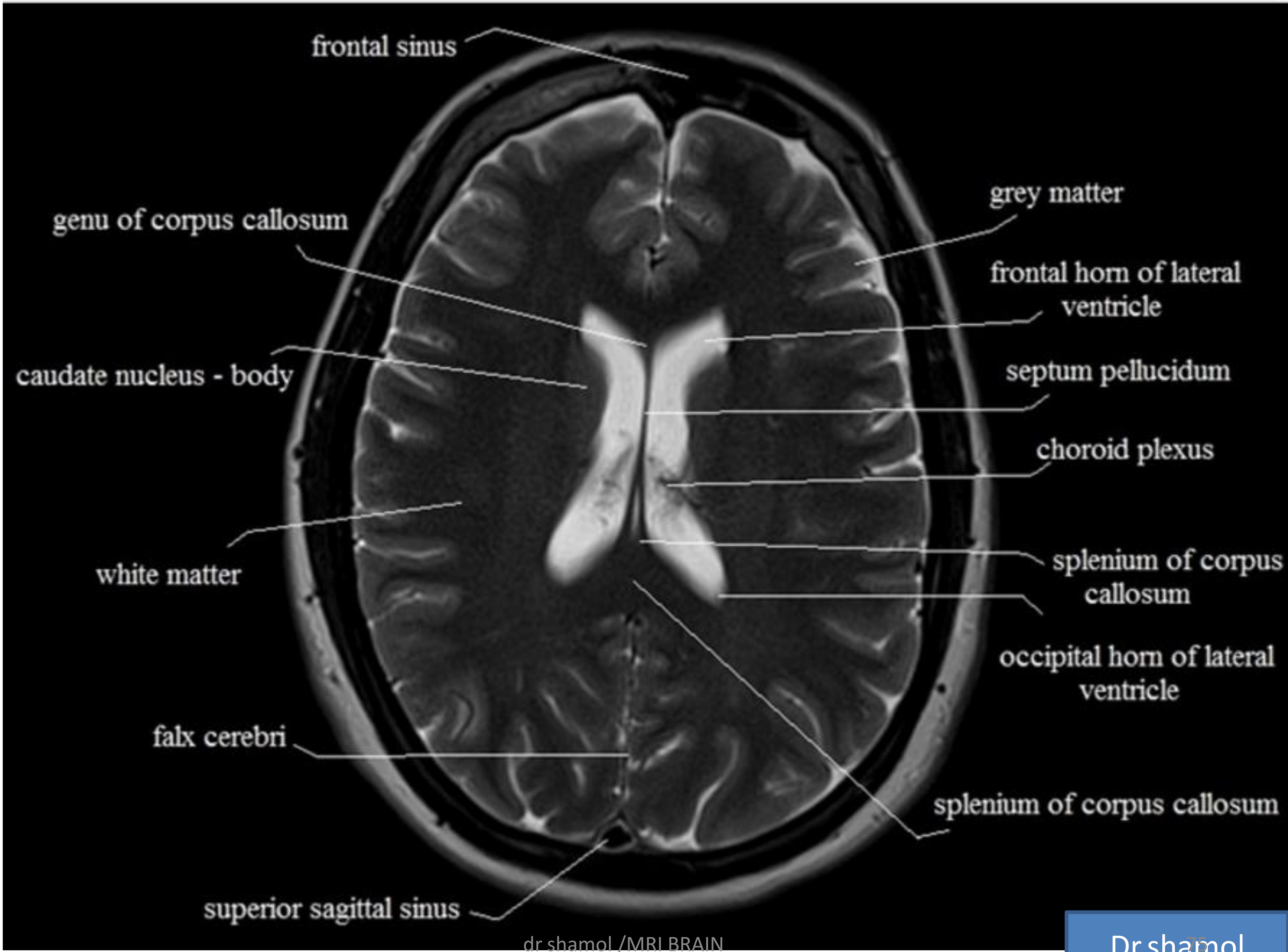
Dr.shamol

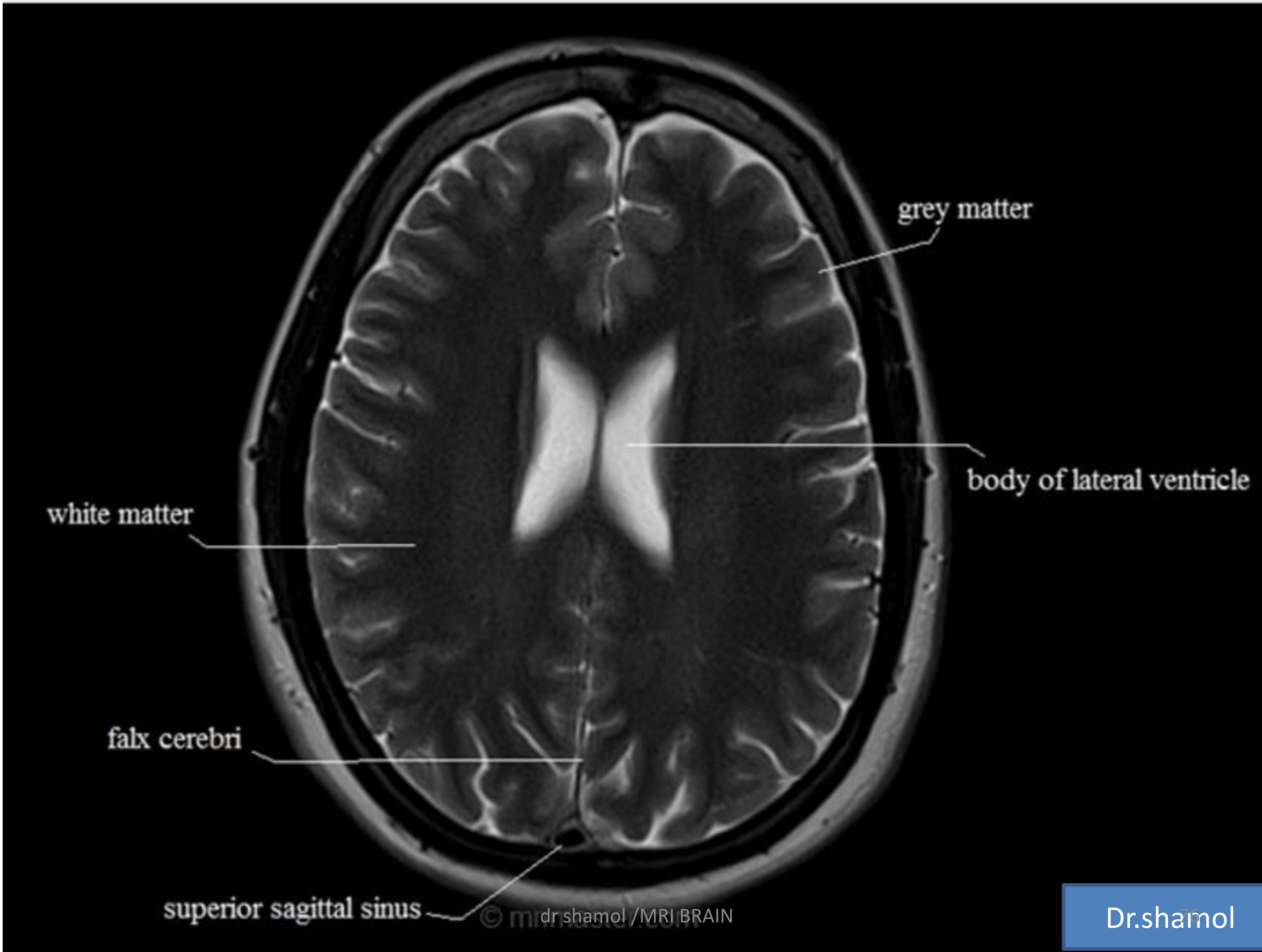

frontal sinus
grey matter
anterior cerebral arteries
caudate nucleus-head
frontal horn of lateral ventricle
interventricular foramen
putamen
globus pallidus
internal cerebral vein
thalamus
white matter
internal cerebral vein
choroid plexus
vein of galen
occipital horn of lateral ventricle
falx cerebri
straight sinus
superior sagittal sinus
© mrimaster.com

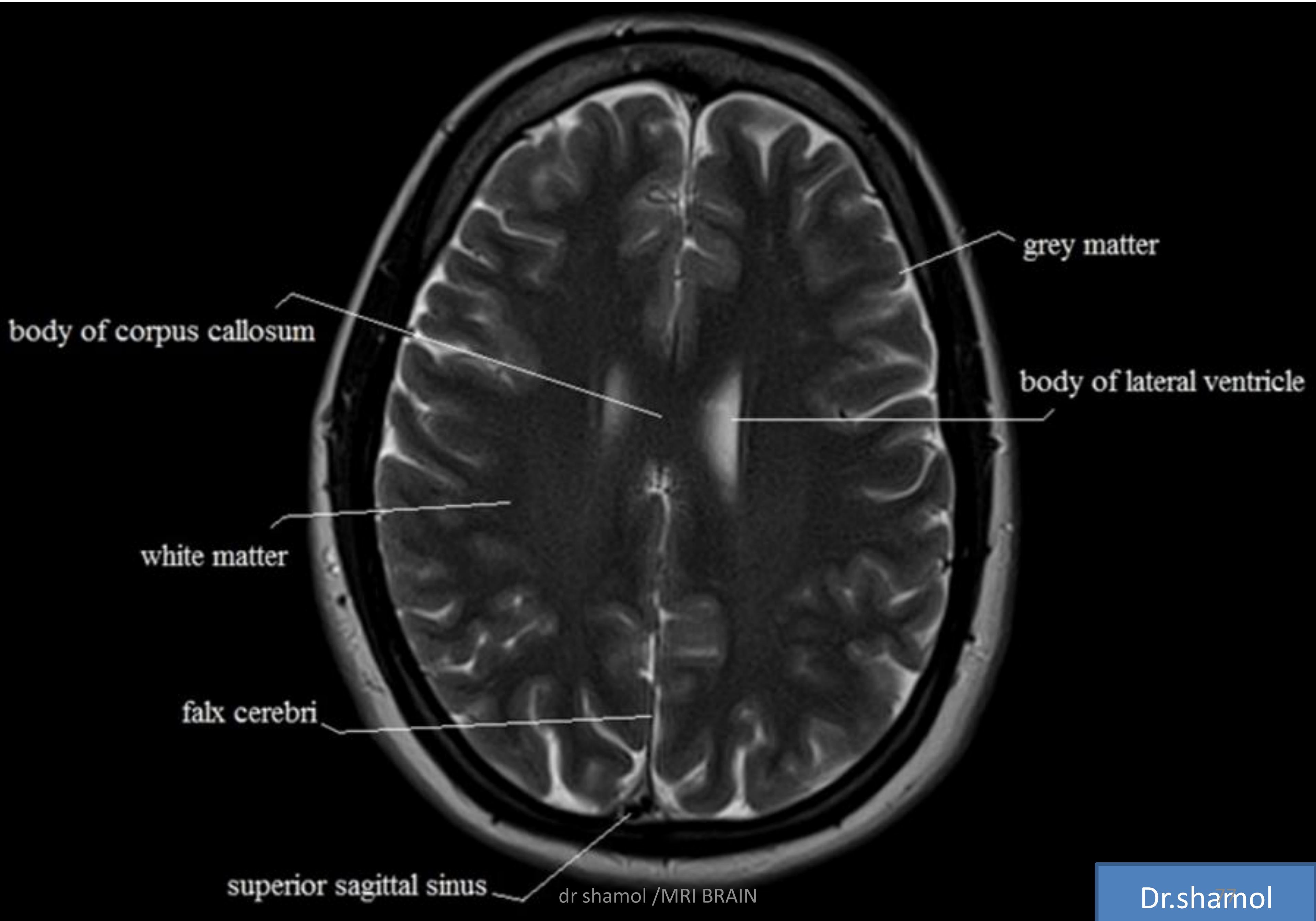

frontal sinus
grey matter
anterior cerebral arteries
caudate nucleus-head
frontal horn of lateral ventricle
interventricular foramen
putamen
globus pallidus
internal cerebral vein
thalamus
white matter
internal cerebral vein
choroid plexus
vein of galen
occipital horn of lateral ventricle
falx cerebri
straight sinus
superior sagittal sinus
© mrimaster.com


frontal sinus
corpus callosum
grey matter
caudate nucleus-head
frontal horn of lateral ventricle
internal cerebral vein
putamen
choroidal vessels
thalamus
internal cerebral vein
white matter
choroid plexus
occipital horn of lateral ventricle
falx cerebri
splenium of corpus callosum
superior sagittal sinus
straight sinus

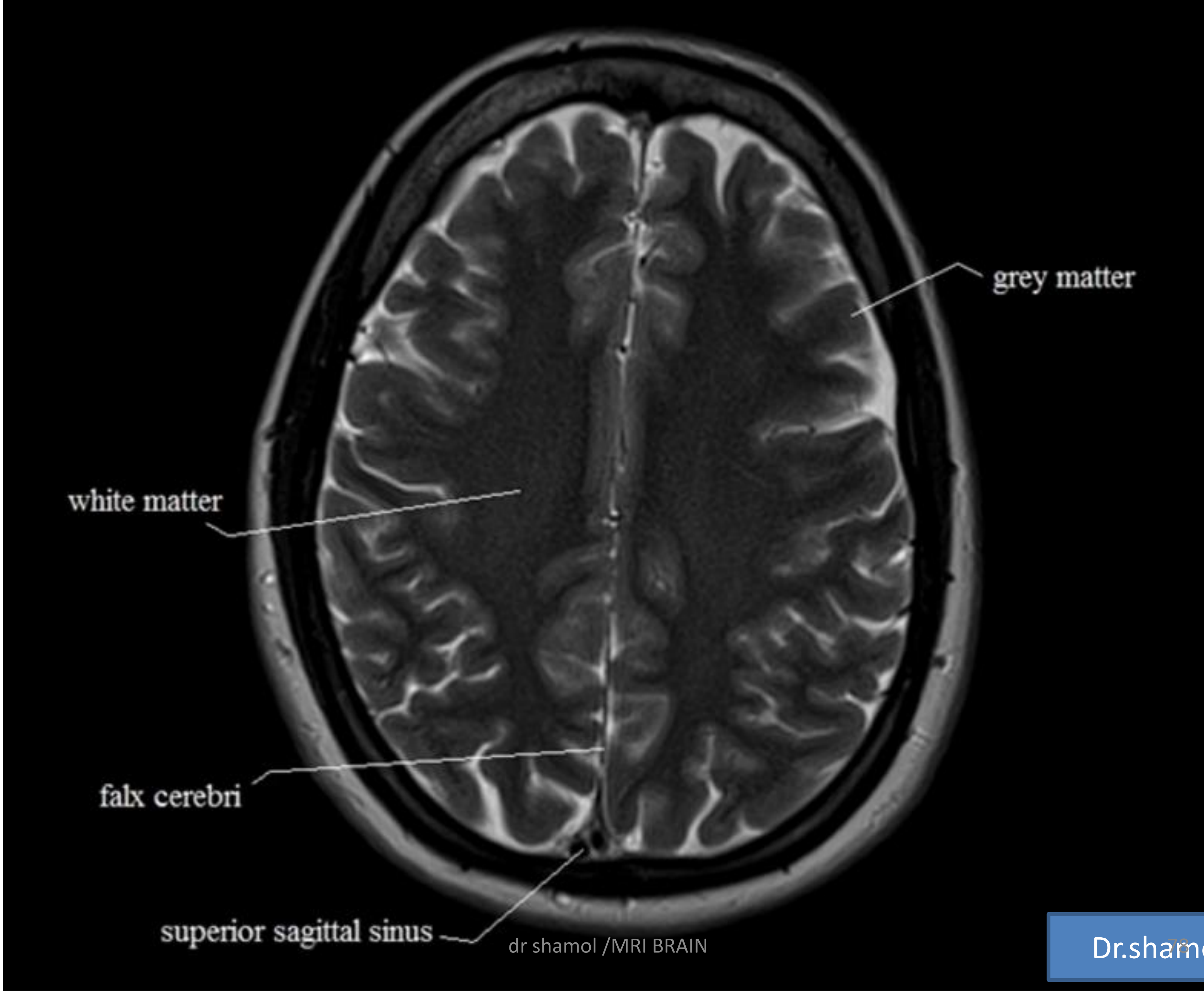

frontal sinus
genu of corpus callosum
caudate nucleus-head
white matter
falx cerebri
superior sagittal sinus
grey matter
frontal horn of lateral ventricle
septum pellucidum
putamen
thalamus
choroid plexus
occipital horn of lateral ventricle
splenium of corpus callosum
© mrimaster.com

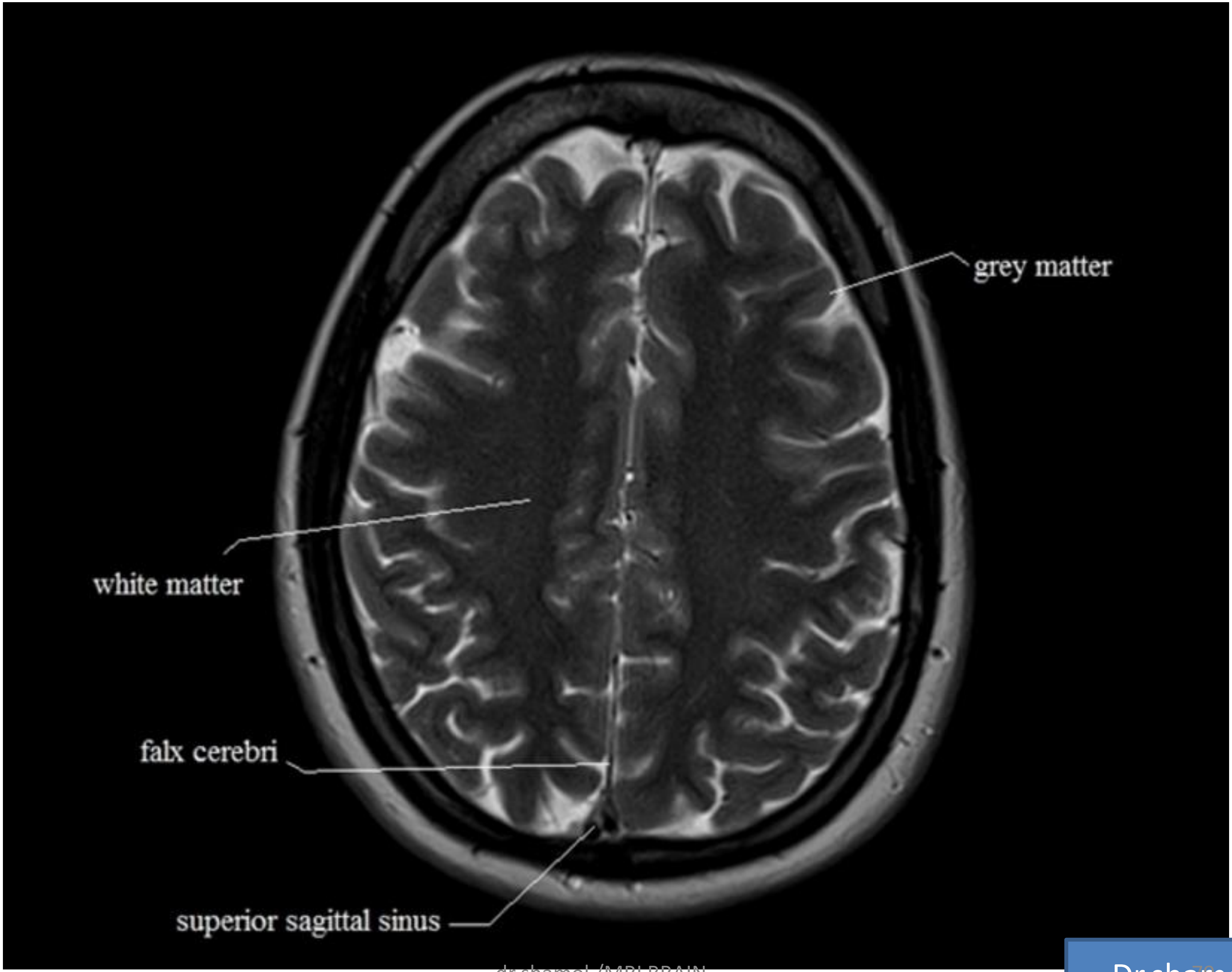

frontal sinus
grey matter
genu of corpus callosum
frontal horn of lateral ventricle
caudate nucleus - body
septum pellucidum
choroid plexus
white matter
splenium of corpus callosum
occipital horn of lateral ventricle
falx cerebri
splenium of corpus callosum
superior sagittal sinus

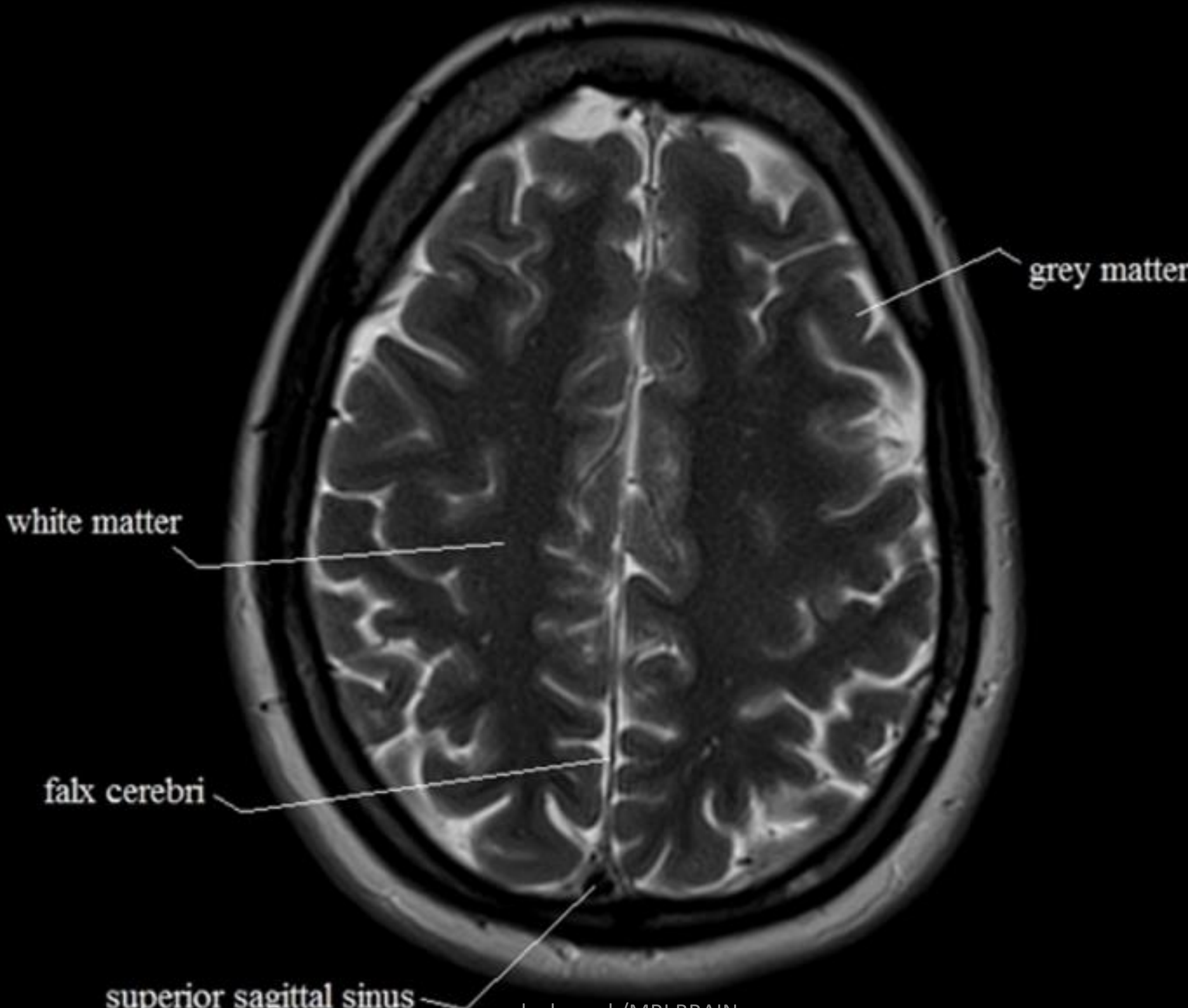

grey matter
body of lateral ventricle
white matter
falx cerebri
superior sagittal sinus

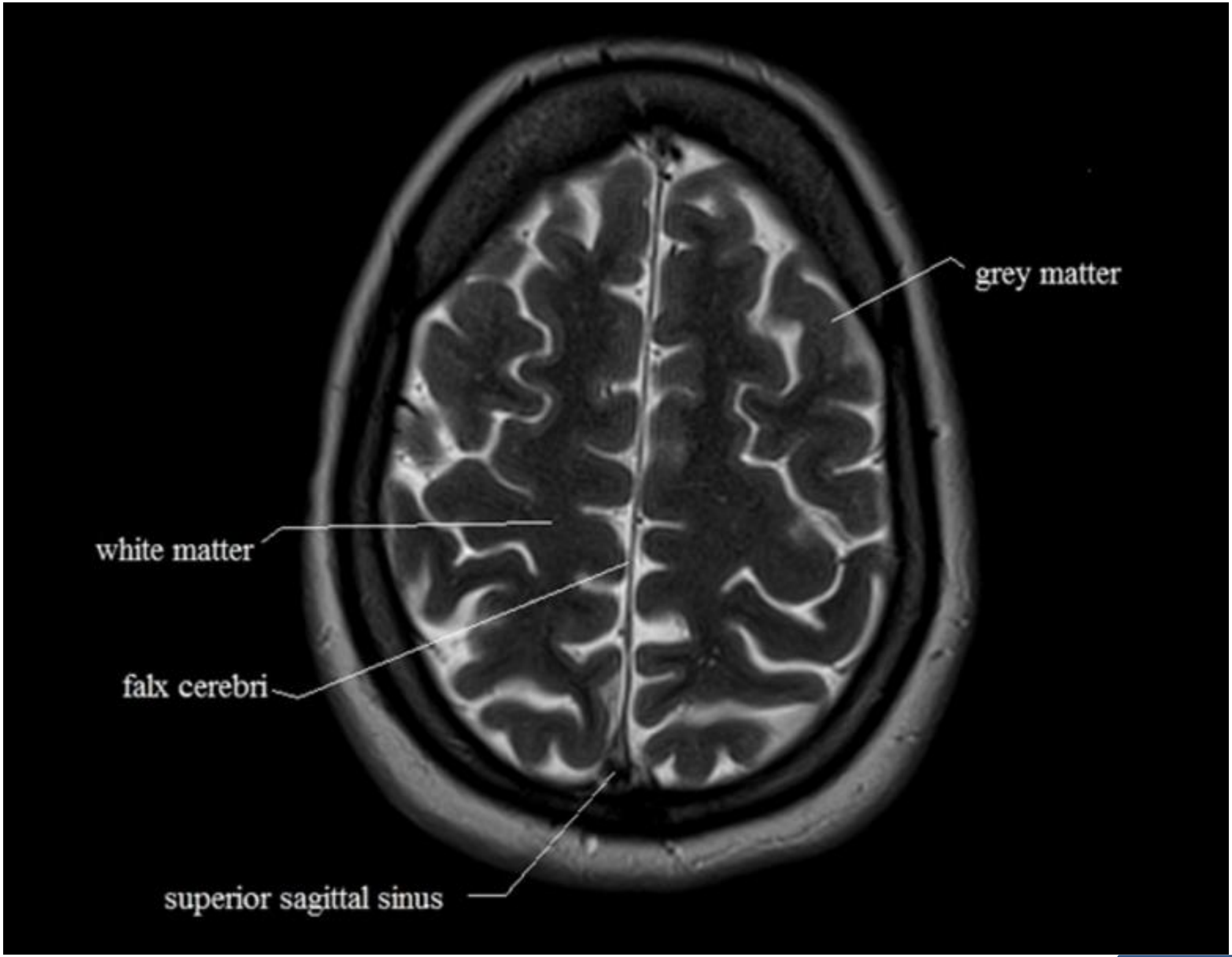

grey matter
body of corpus callosum
body of lateral ventricle
white matter
falx cerebri
superior sagittal sinus

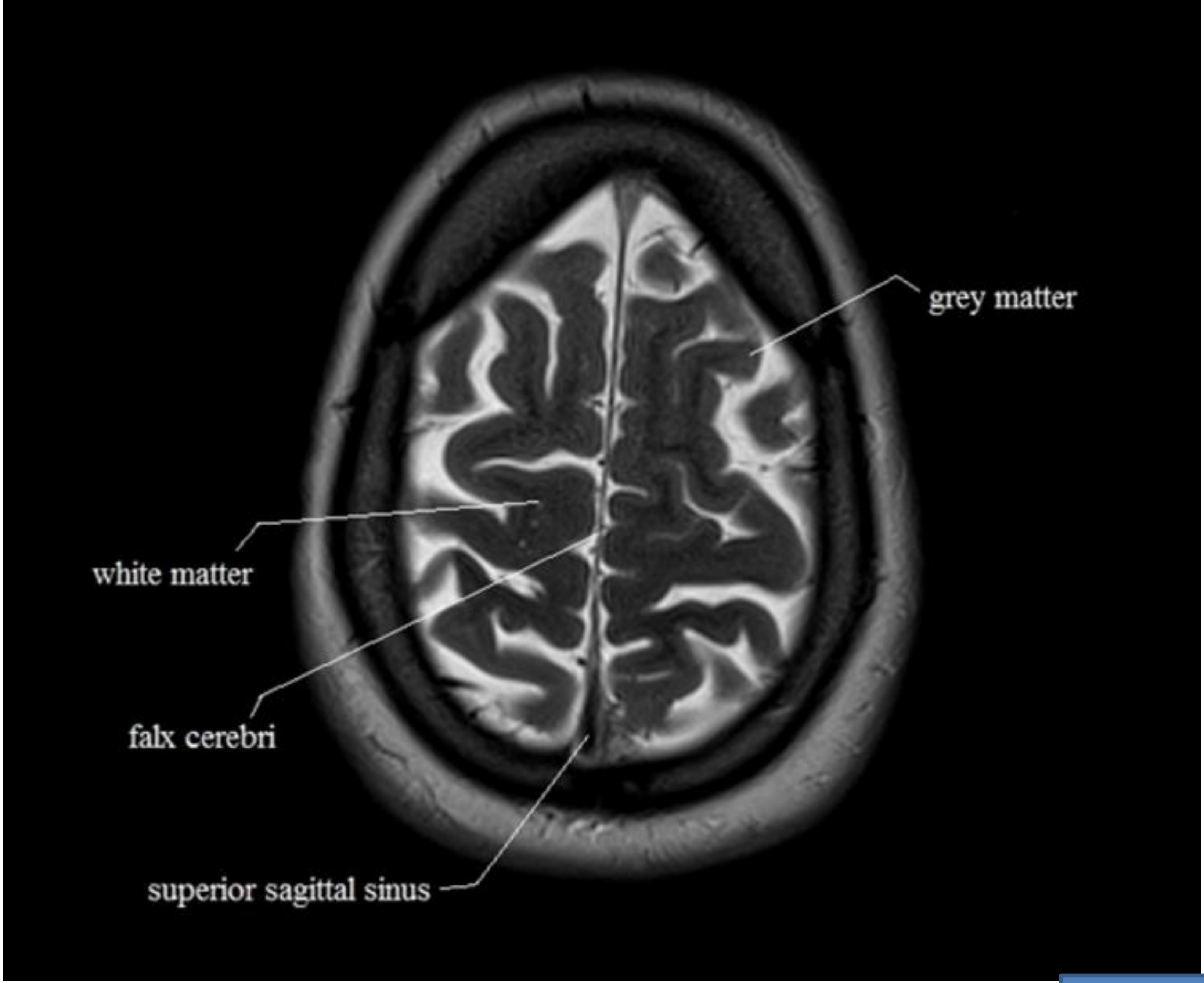
Dr.shamol


grey matter
white matter
falx cerebri
superior sagittal sinus


grey matter
white matter
falx cerebri
superior sagittal sinus


grey matter
white matter
falx cerebri
superior sagittal sinus


grey matter
white matter
falx cerebri
superior sagittal sinus


grey matter
white matter
falx cerebri
superior sagittal sinus

NOW IDENTIFY THE STRUCTURE’S BY YOUR SELF

ROW -1

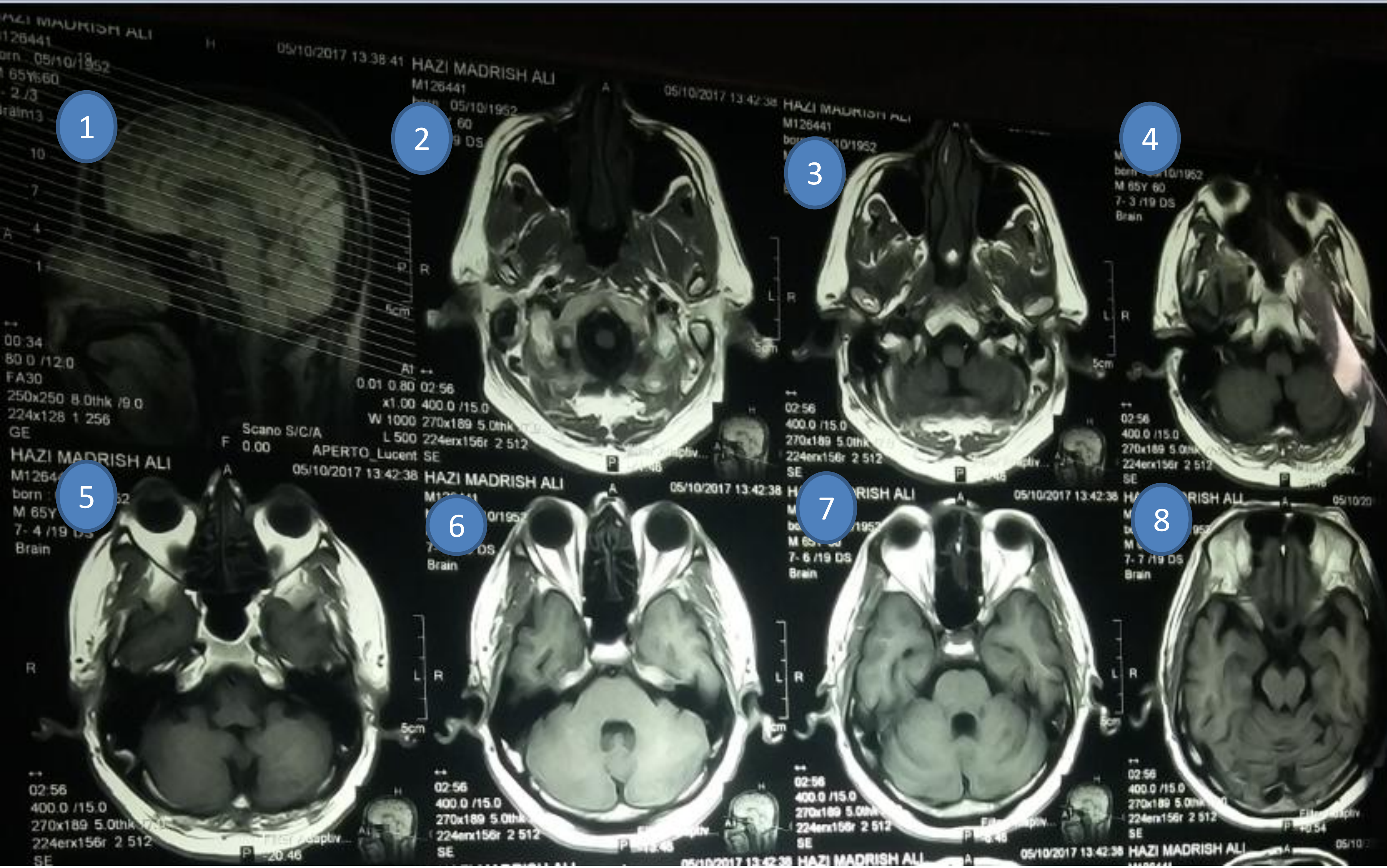
ROW -2

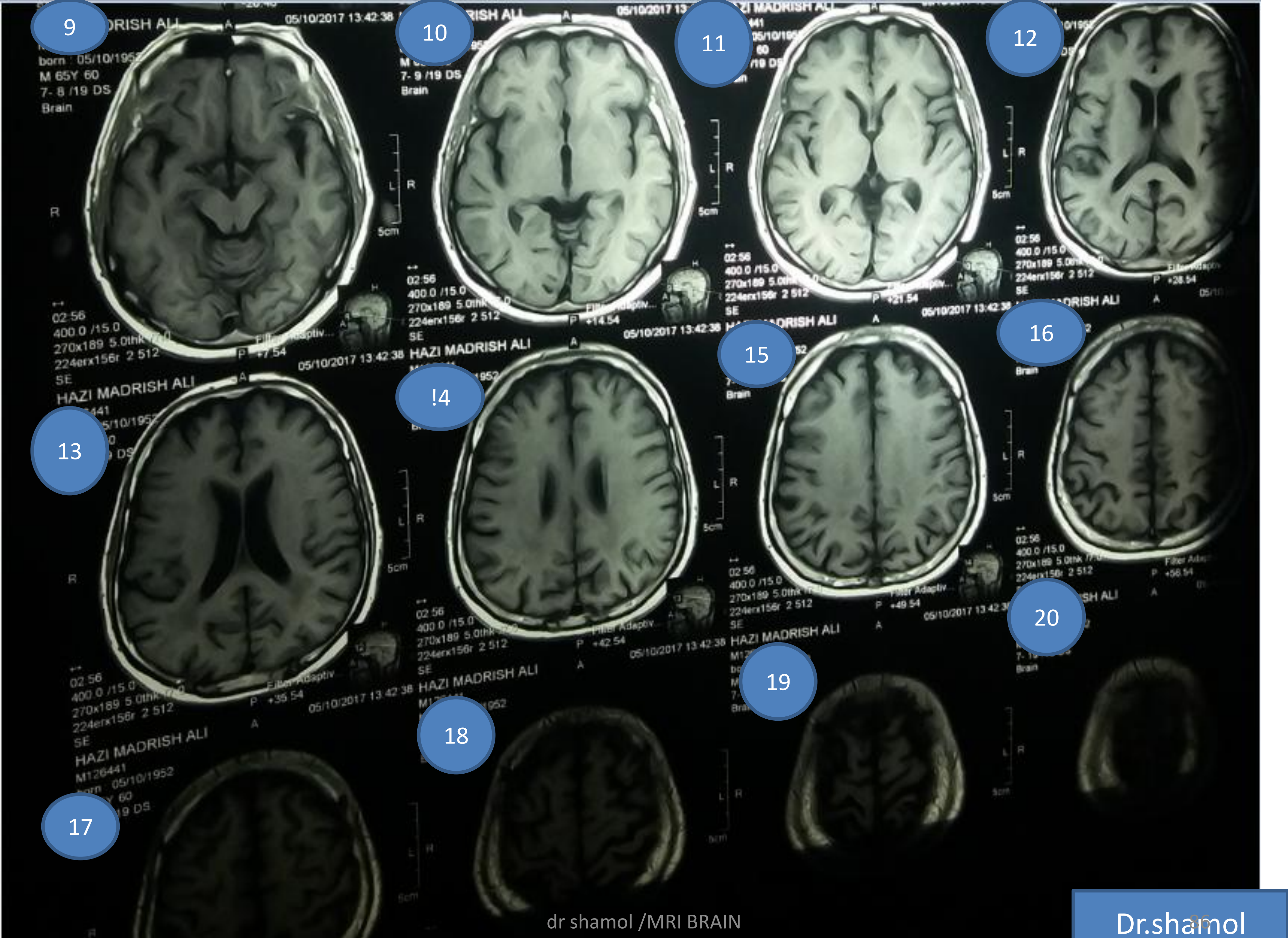
ROW -3

ROW -4

ROW -5

Dr.shamol


1
2
3
4
5
6
7
8
HAZI MADRISH ALI
05/10/2017 13:42:38
M126441
born 05/10/1952
M 65Y 60
Brain
02:56
400.0 /15.0
270x189 5.0thk
224erx156r 2 512
SE
5cm



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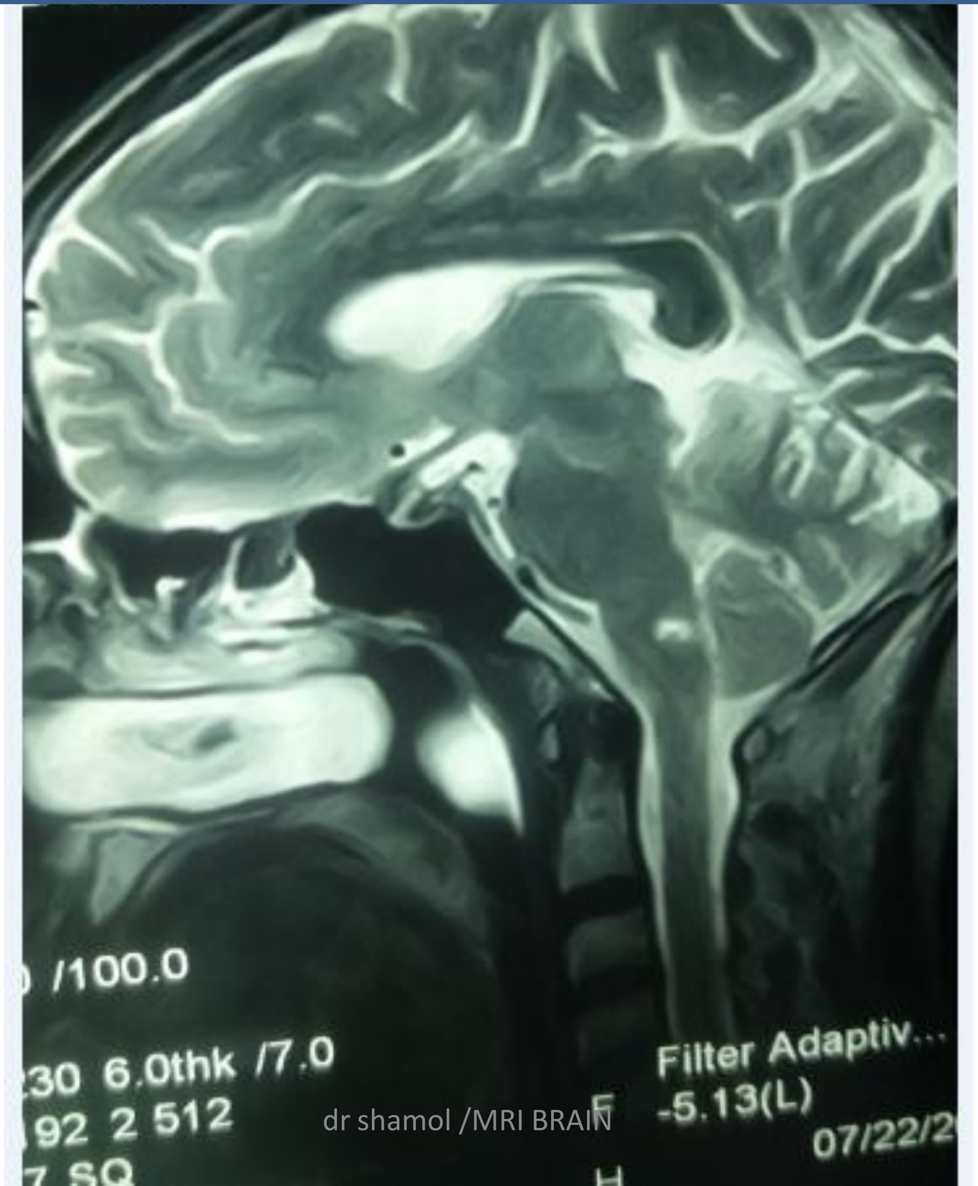
SAGITAL



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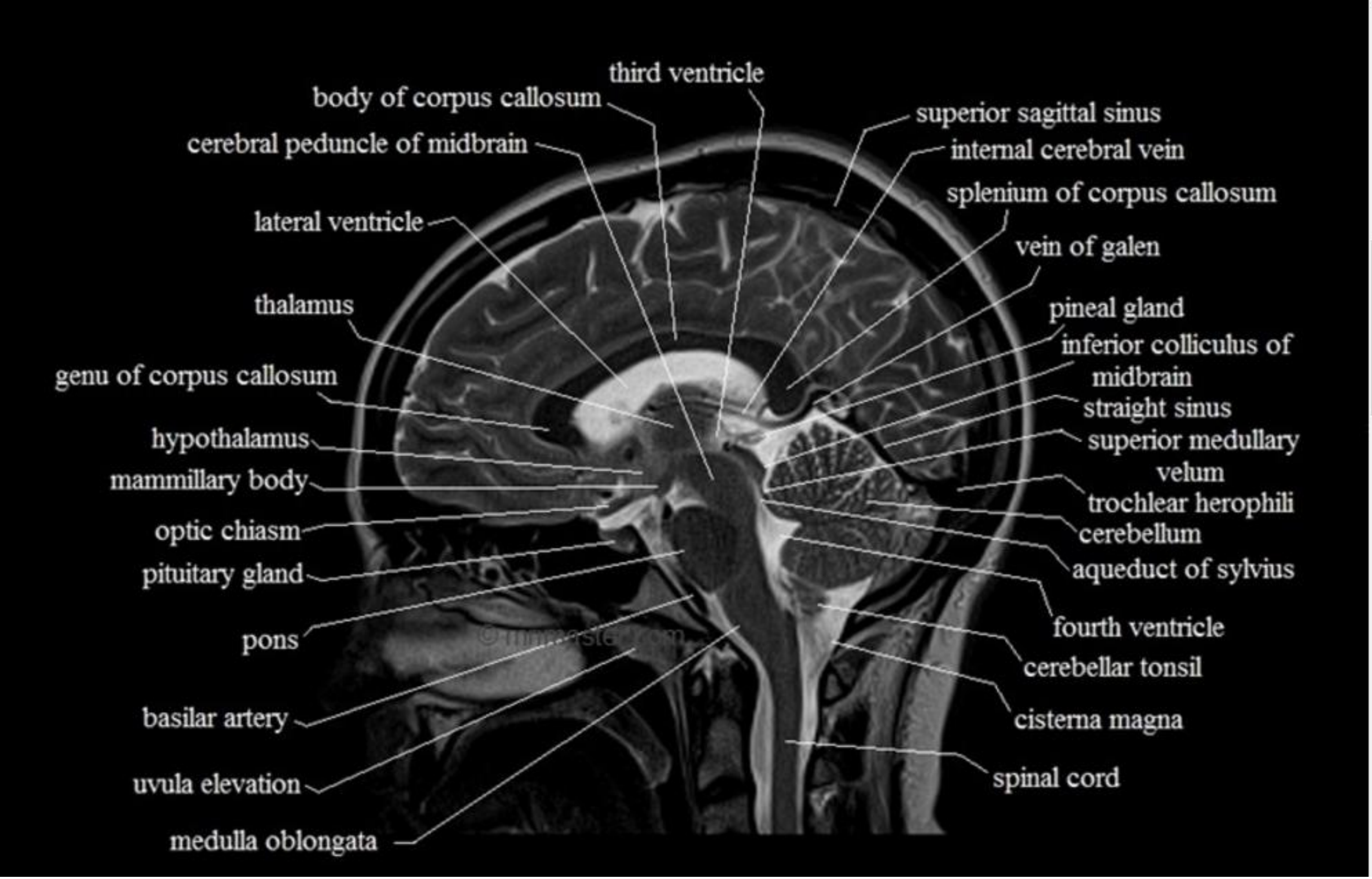
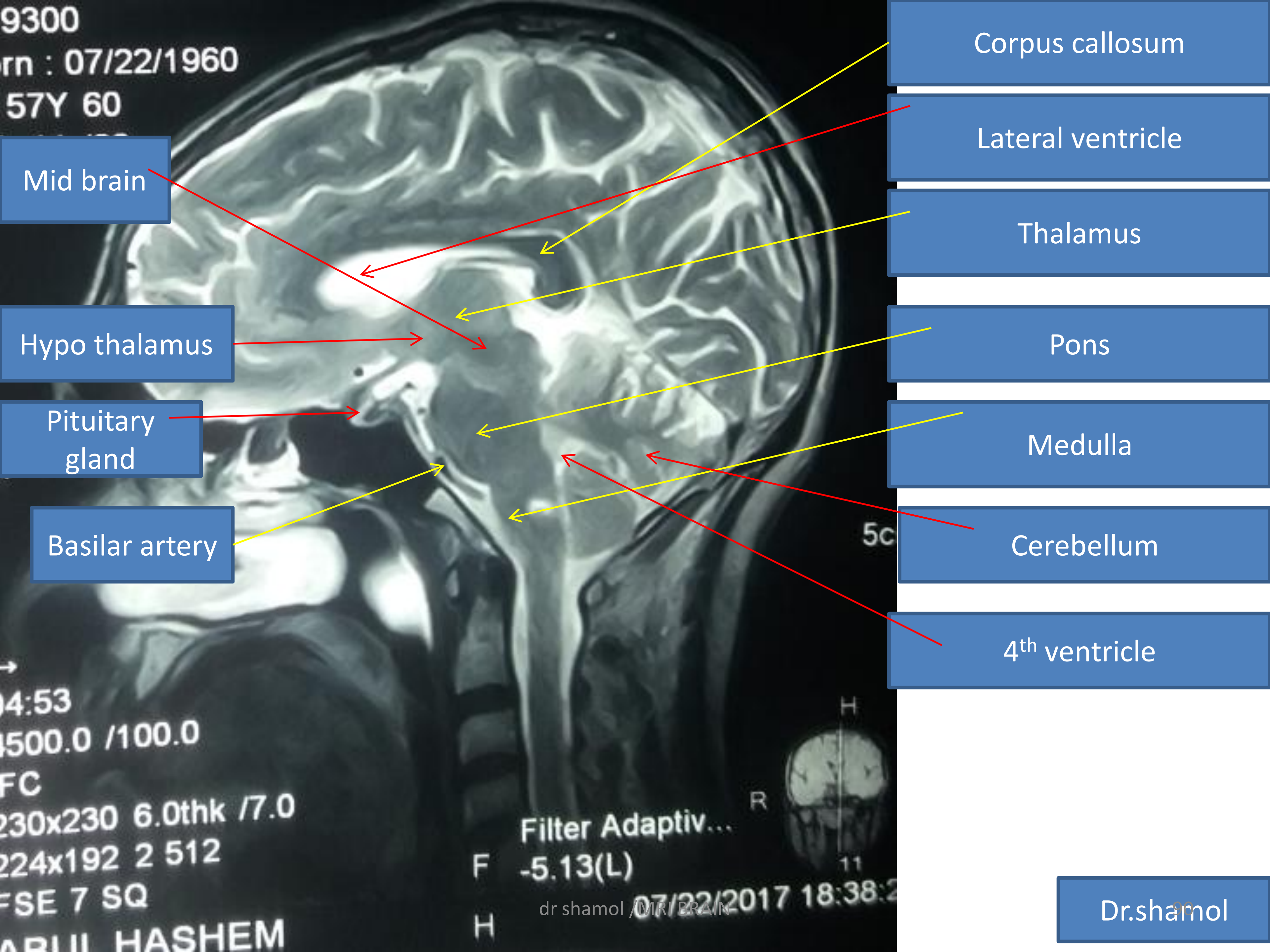
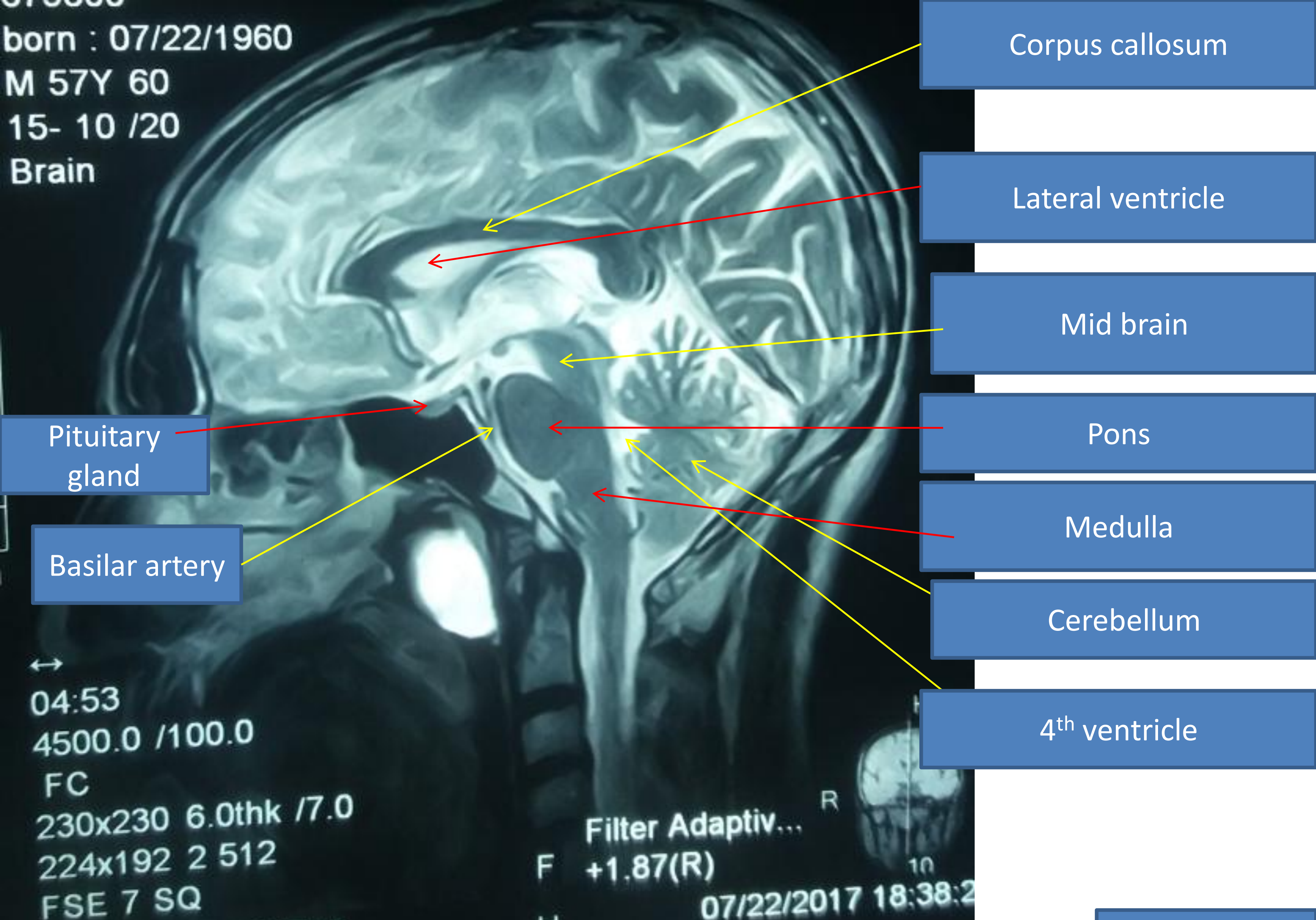
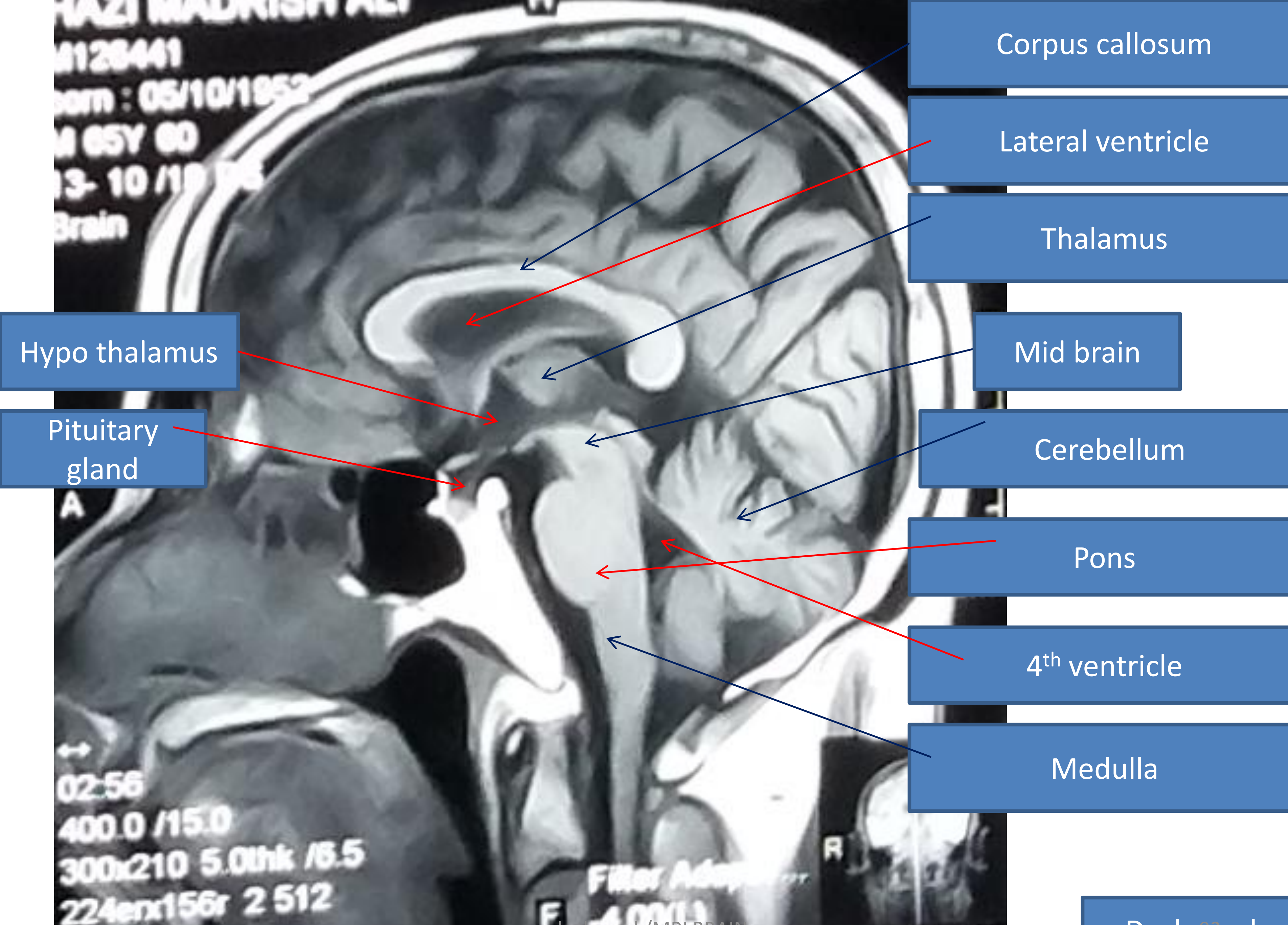
T2

T1

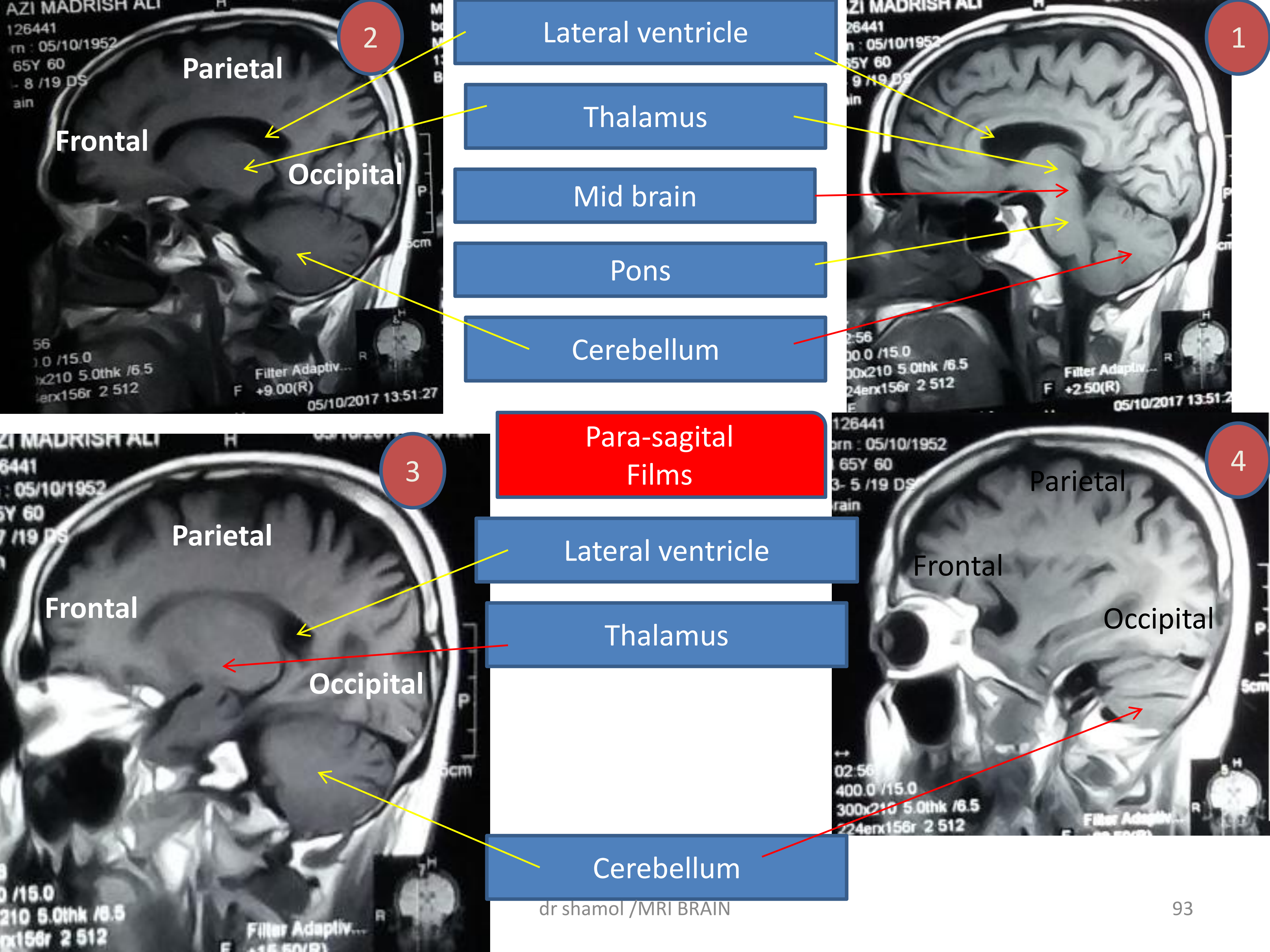

third ventricle
body of corpus callosum
cerebral peduncle of midbrain
superior sagittal sinus
internal cerebral vein
splenium of corpus callosum
lateral ventricle
vein of galen
thalamus
pineal gland
inferior colliculus of midbrain
genu of corpus callosum
straight sinus
hypothalamus
superior medullary velum
mammillary body
trochlear herophili
optic chiasm
cerebellum
pituitary gland
aqueduct of sylvius
pons
fourth ventricle
cerebellar tonsil
basilar artery
cisterna magna
uvula elevation
spinal cord
medulla oblongata


Corpus callosum
Lateral ventricle
Thalamus
Pons
Medulla
Cerebellum
4th ventricle
Mid brain
Hypo thalamus
Pituitary gland
Basilar artery
9300
rn : 07/22/1960
57Y 60
04:53
500.0 /100.0
FC
230x230 6.0thk /7.0
224x192 2 512
FSE 7 SQ
ABUL HASHEM
5c
H
R
11
Filter Adaptiv...
F -5.13(L)
07/22/2017 18:38:2
H
Dr.shamol


born : 07/22/1960
M 57Y 60
15- 10 /20
Brain
Corpus callosum
Lateral ventricle
Mid brain
Pons
Medulla
Cerebellum
4th ventricle
Pituitary gland
Basilar artery
04:53
4500.0 /100.0
FC
230x230 6.0thk /7.0
224x192 2 512
FSE 7 SQ
ABUL HASHEM
Filter Adaptiv...
R
F +1.87(R)
07/22/2017 18:38:2
H
Dr.shamol

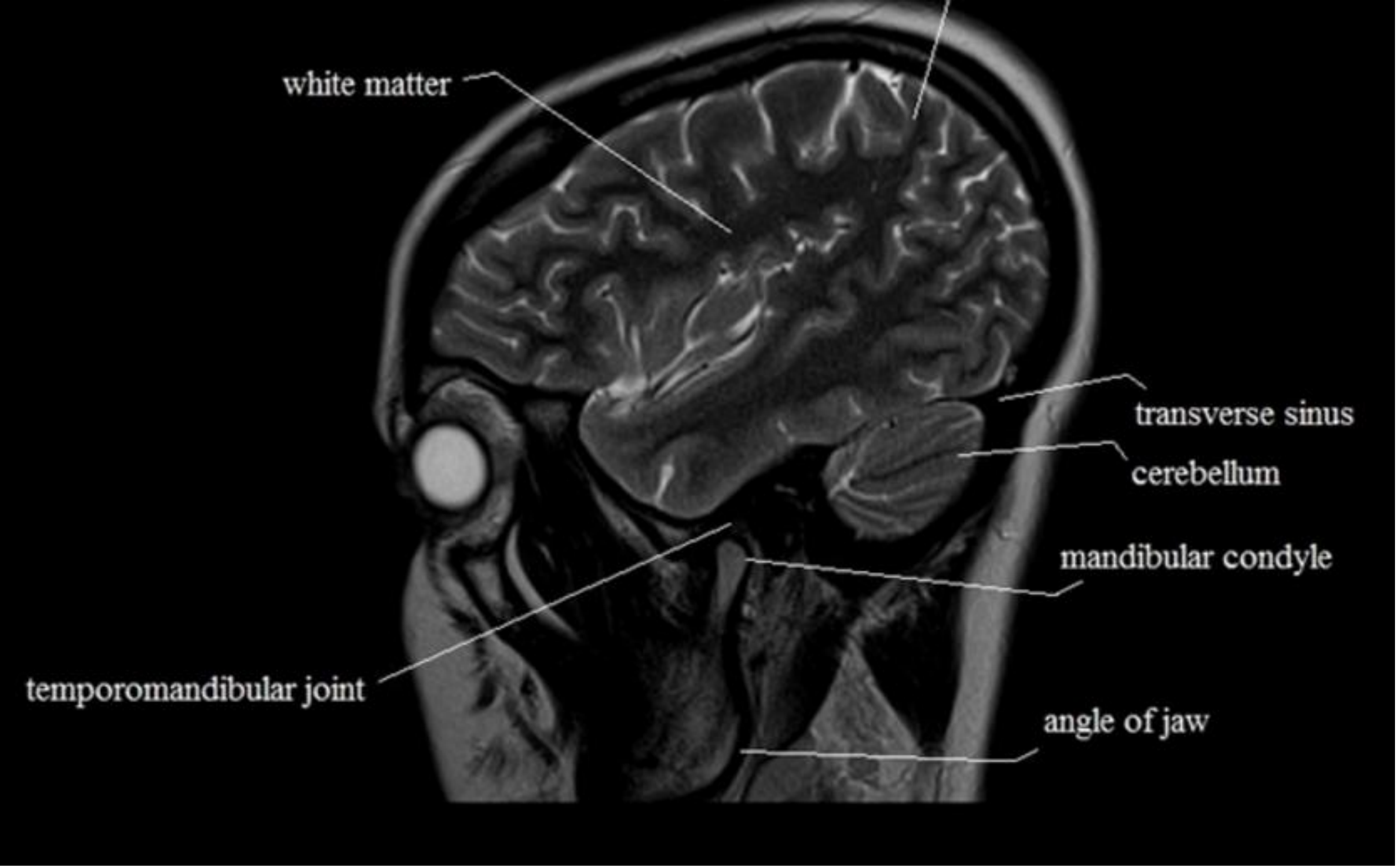

Corpus callosum
Lateral ventricle
Thalamus
Hypo thalamus
Pituitary gland
Mid brain
Cerebellum
Pons
4th ventricle
Medulla
A
R
02:56
400.0 /15.0
300x210 5.0thk /6.5
224erx156r 2 512

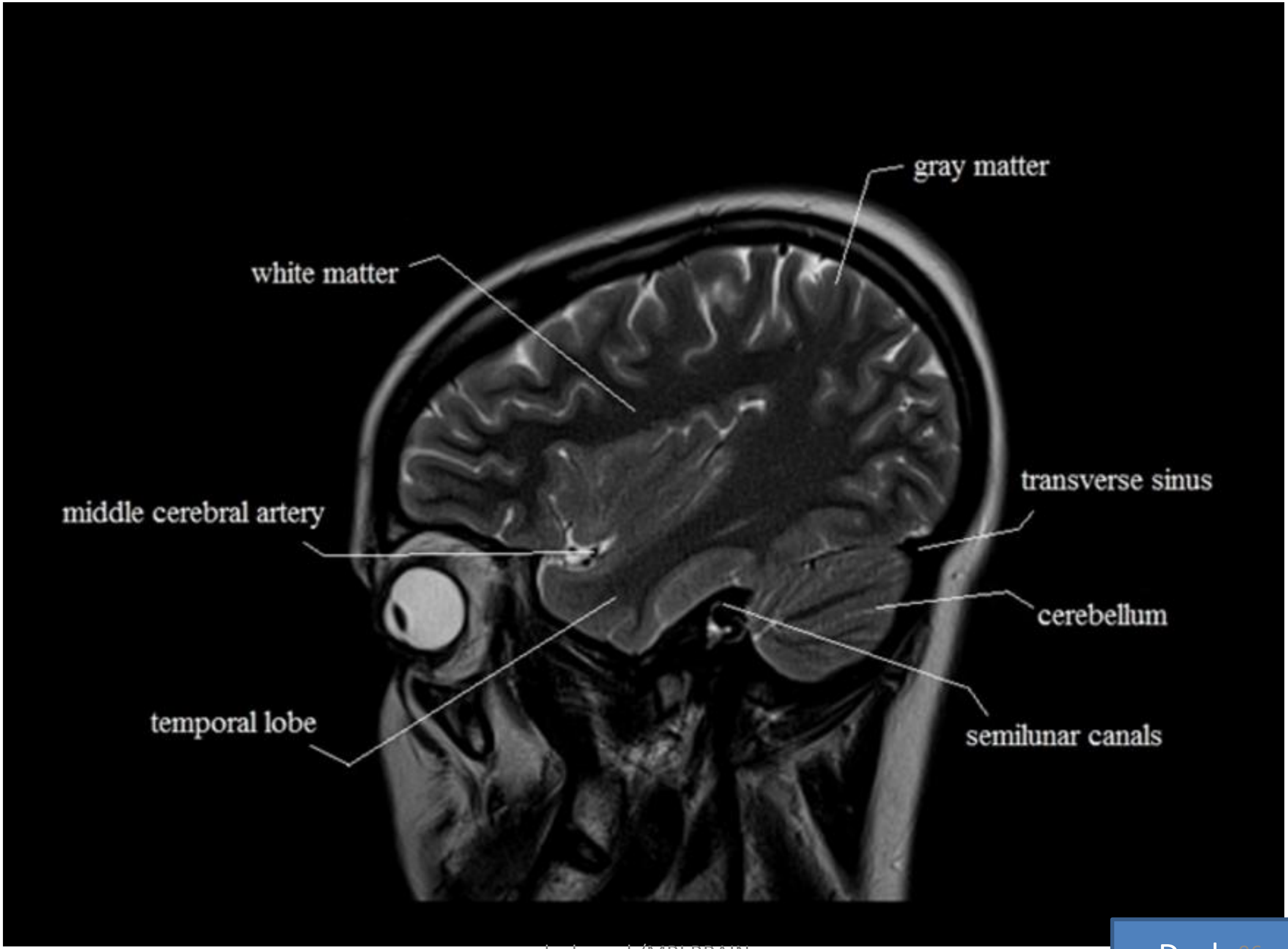
Dr.shamol

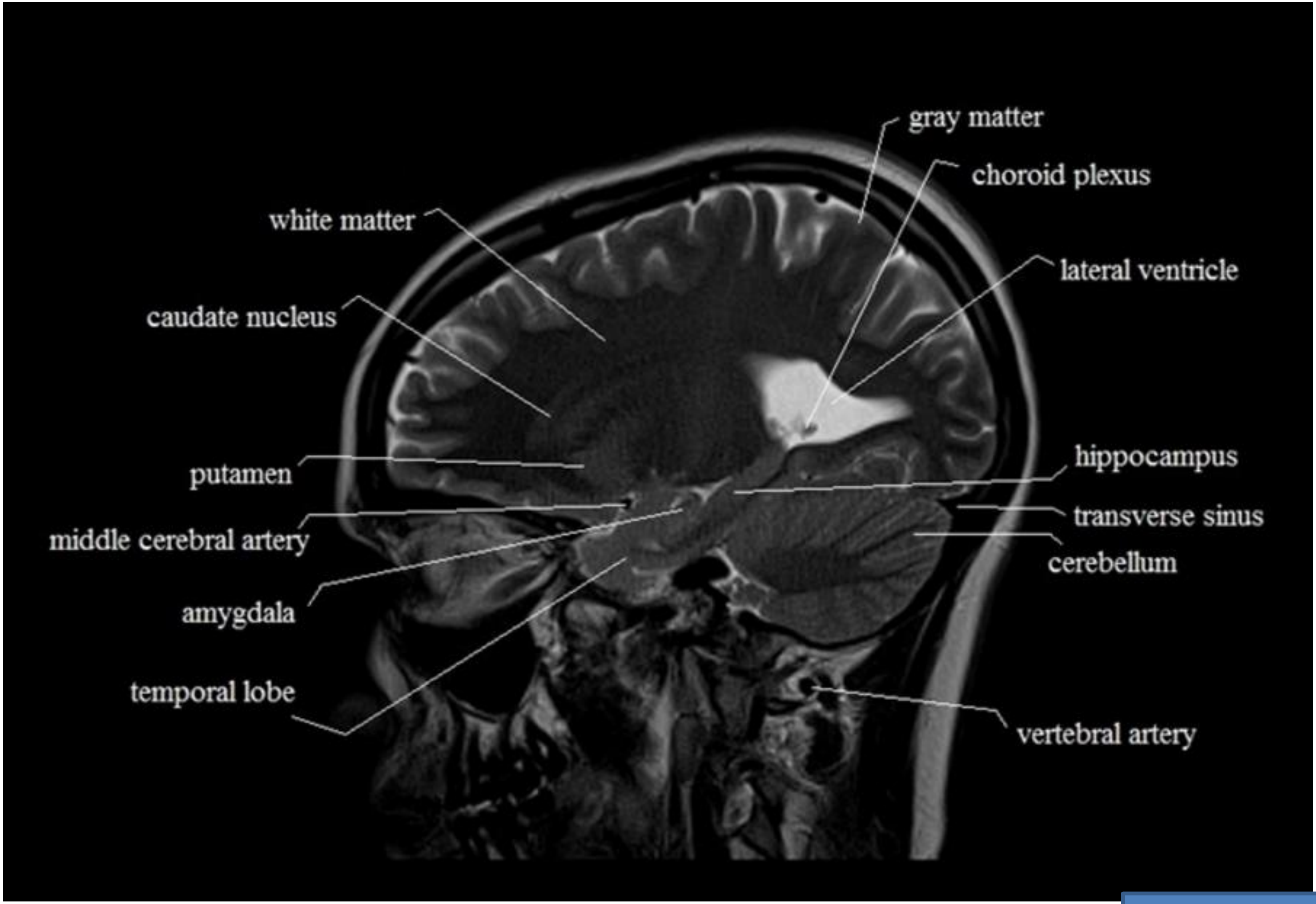

Para-sagital Films
Lateral ventricle
Thalamus
Mid brain
Pons
Cerebellum
Lateral ventricle
Thalamus
Cerebellum
Parietal
Frontal
Occipital
1
2
3
4

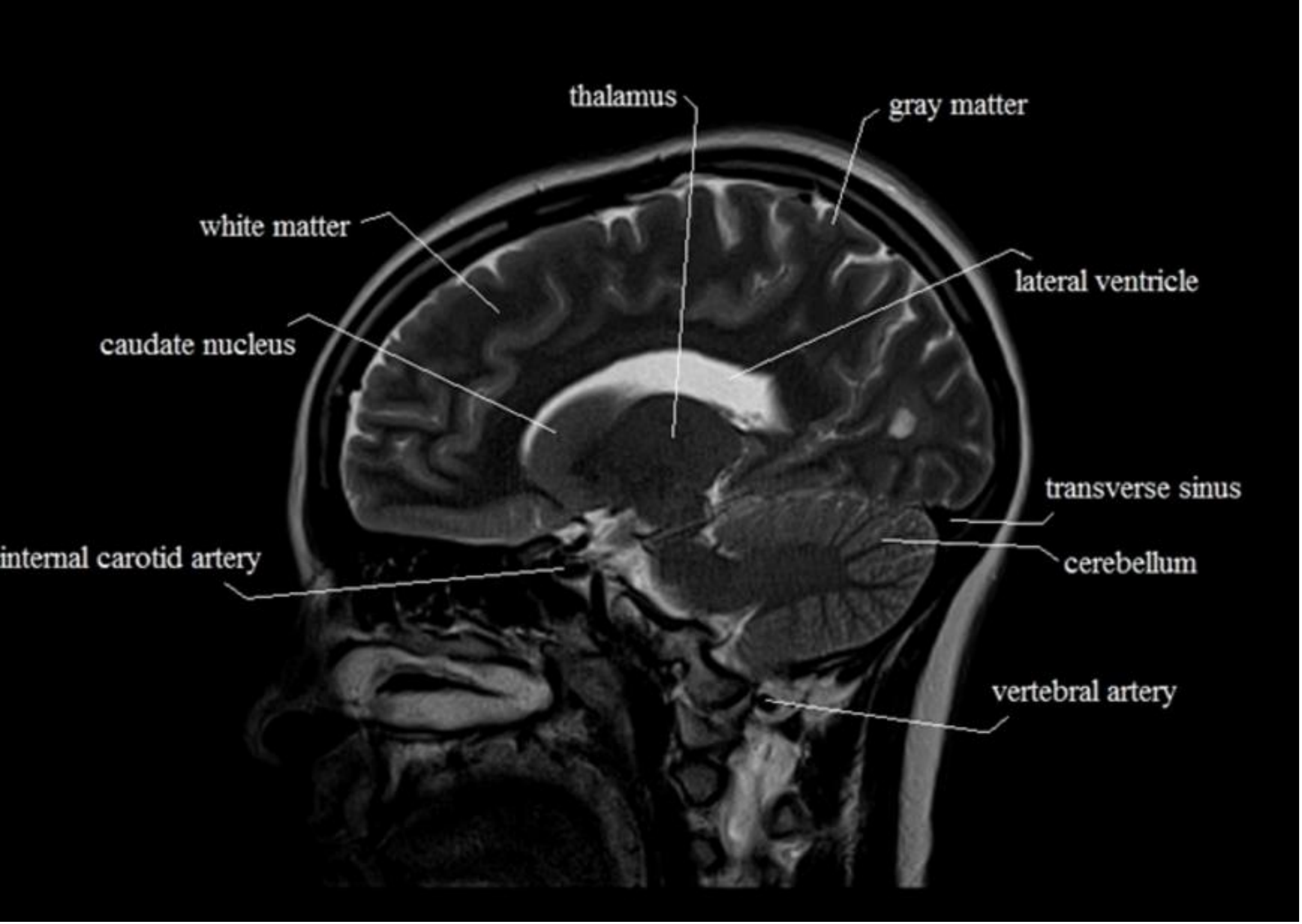
SAGITAL
Structure A to Z

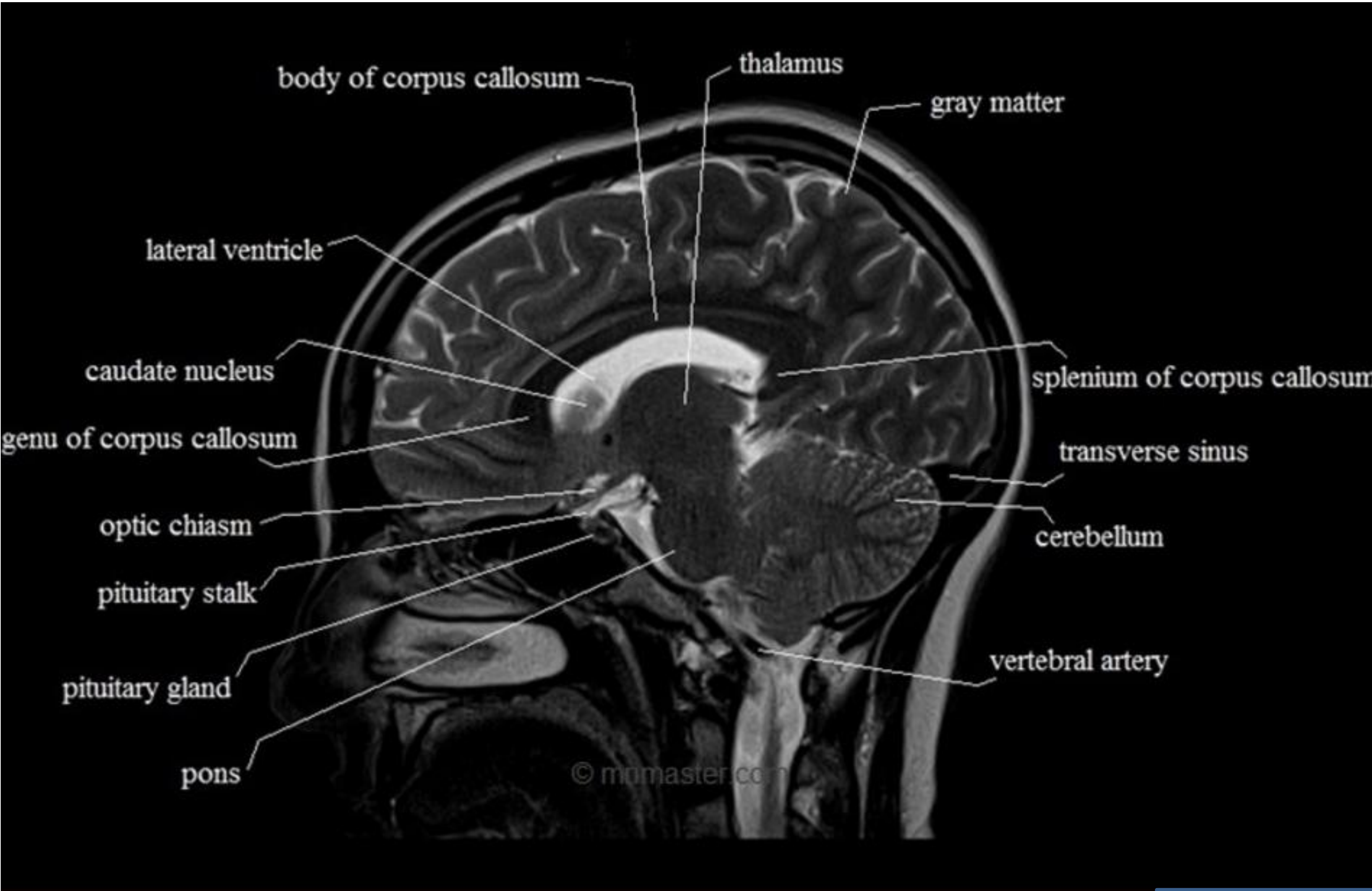
Sequence
FROM Left to → right side

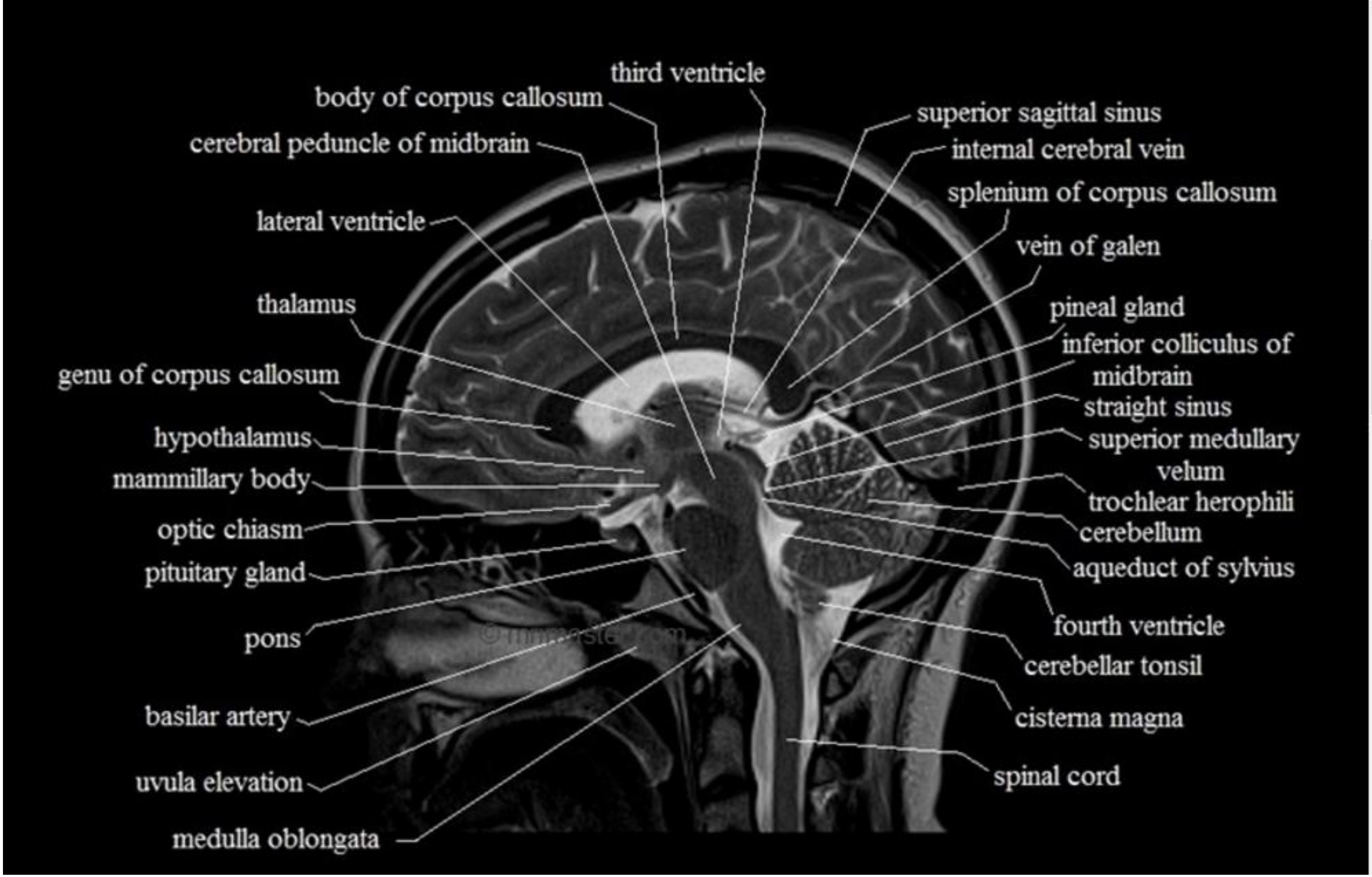

white matter
transverse sinus
cerebellum
mandibular condyle
temporomandibular joint
angle of jaw

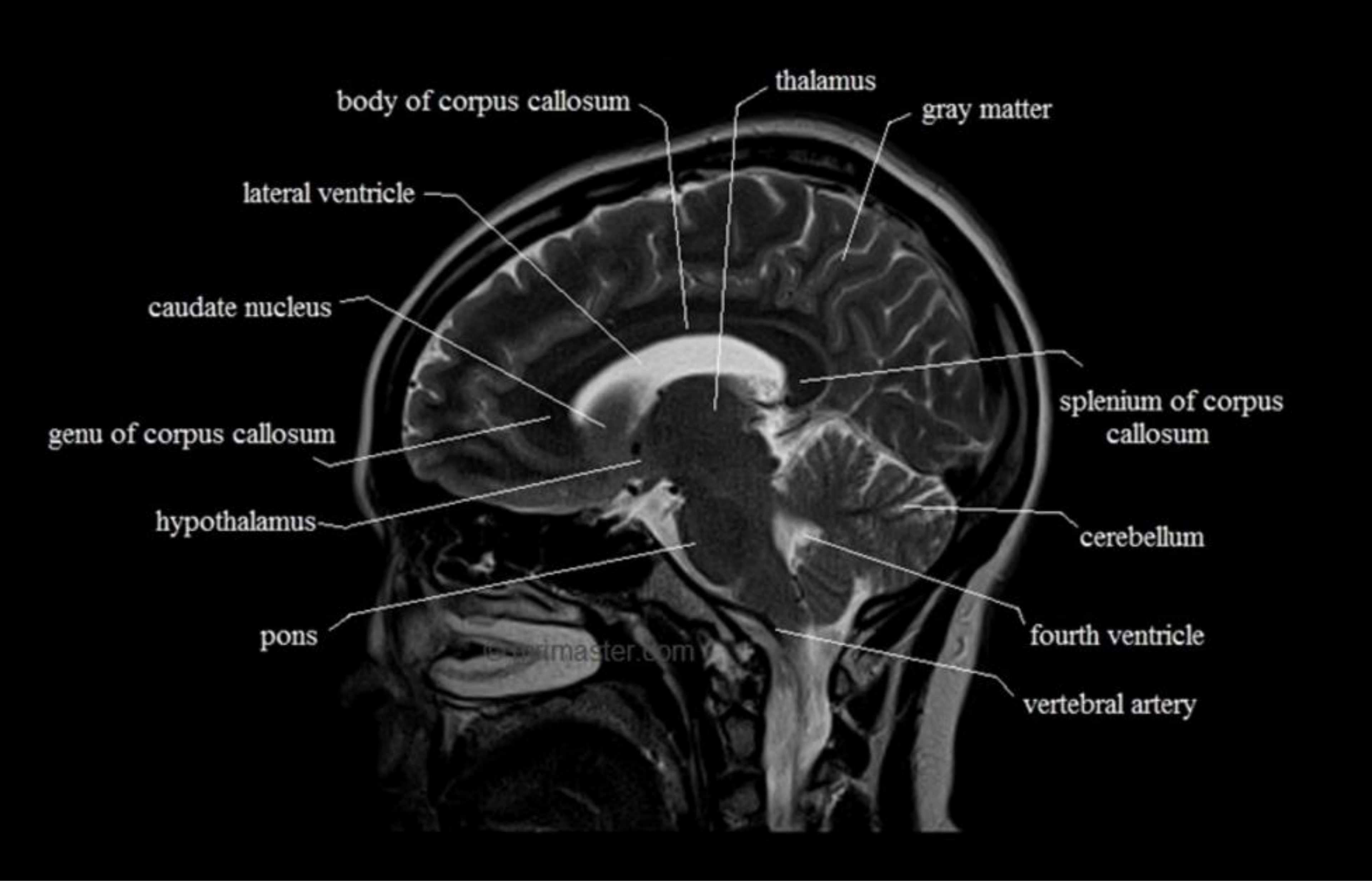

gray matter
white matter
transverse sinus
middle cerebral artery
cerebellum
temporal lobe
semilunar canals

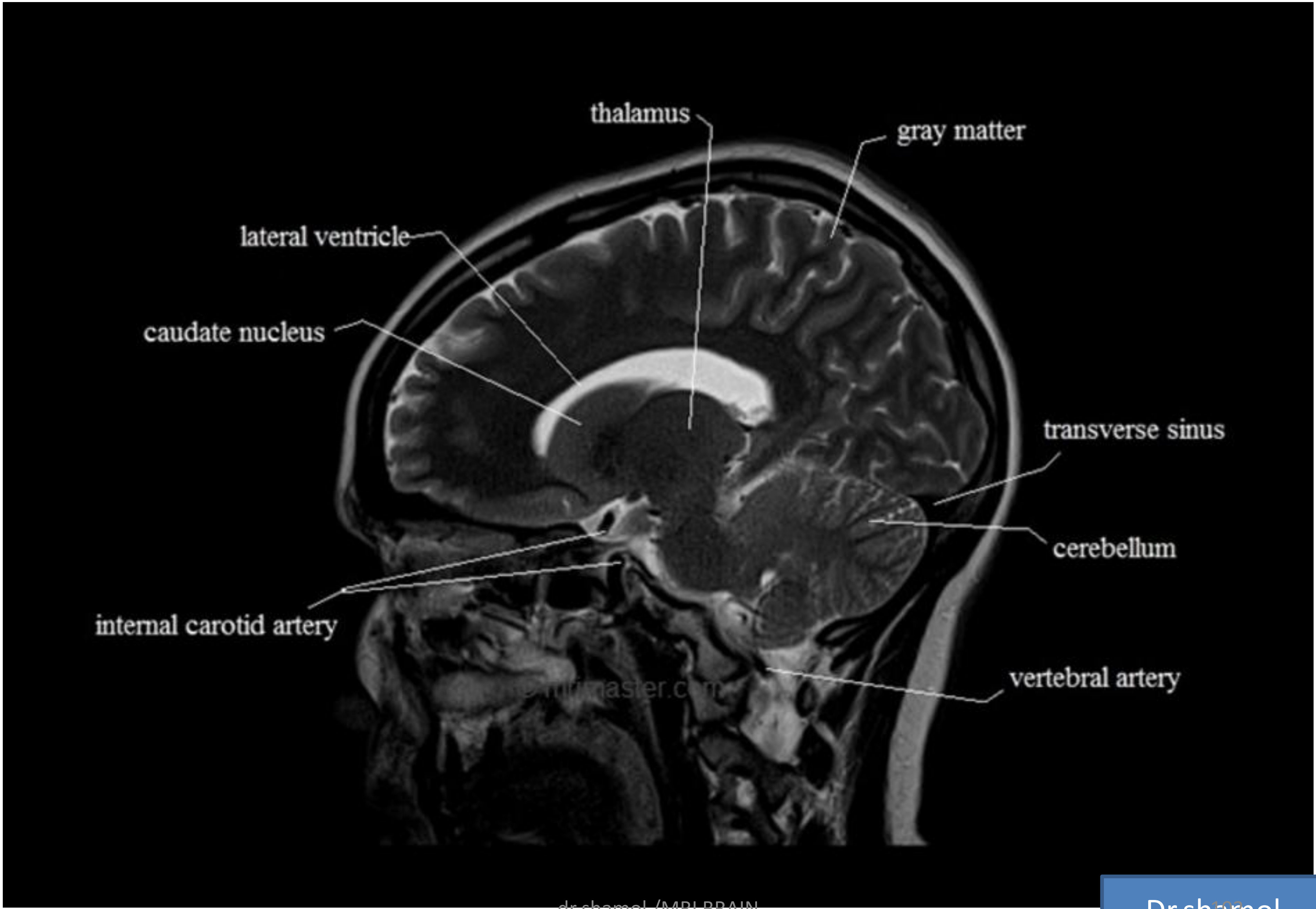

gray matter
choroid plexus
white matter
lateral ventricle
caudate nucleus
putamen
hippocampus
middle cerebral artery
transverse sinus
cerebellum
amygdala
temporal lobe
vertebral artery


thalamus
gray matter
white matter
lateral ventricle
caudate nucleus
transverse sinus
internal carotid artery
cerebellum
vertebral artery

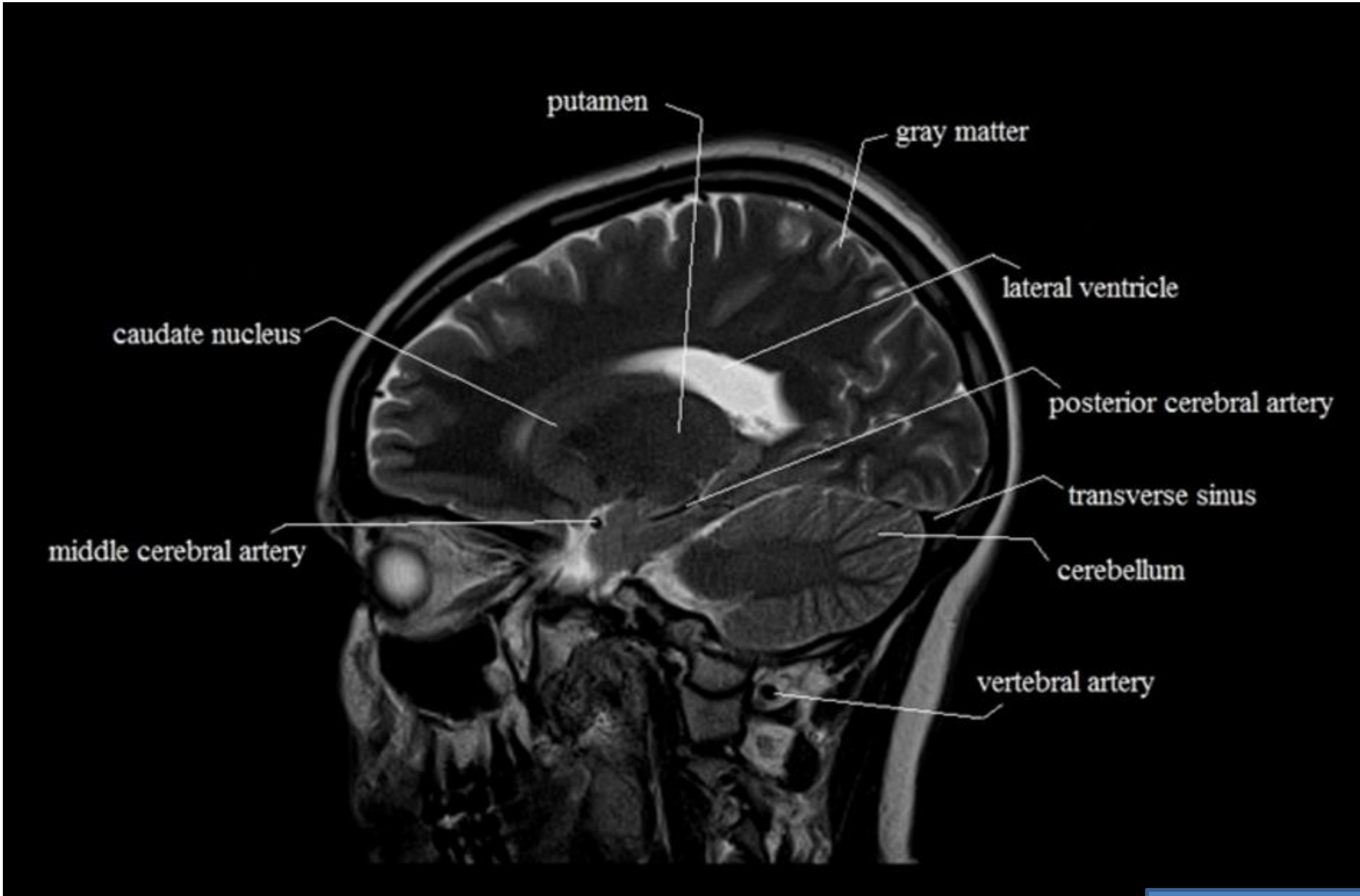

body of corpus callosum
thalamus
gray matter
lateral ventricle
caudate nucleus
splenium of corpus callosum
genu of corpus callosum
transverse sinus
optic chiasm
cerebellum
pituitary stalk
pituitary gland
vertebral artery
pons

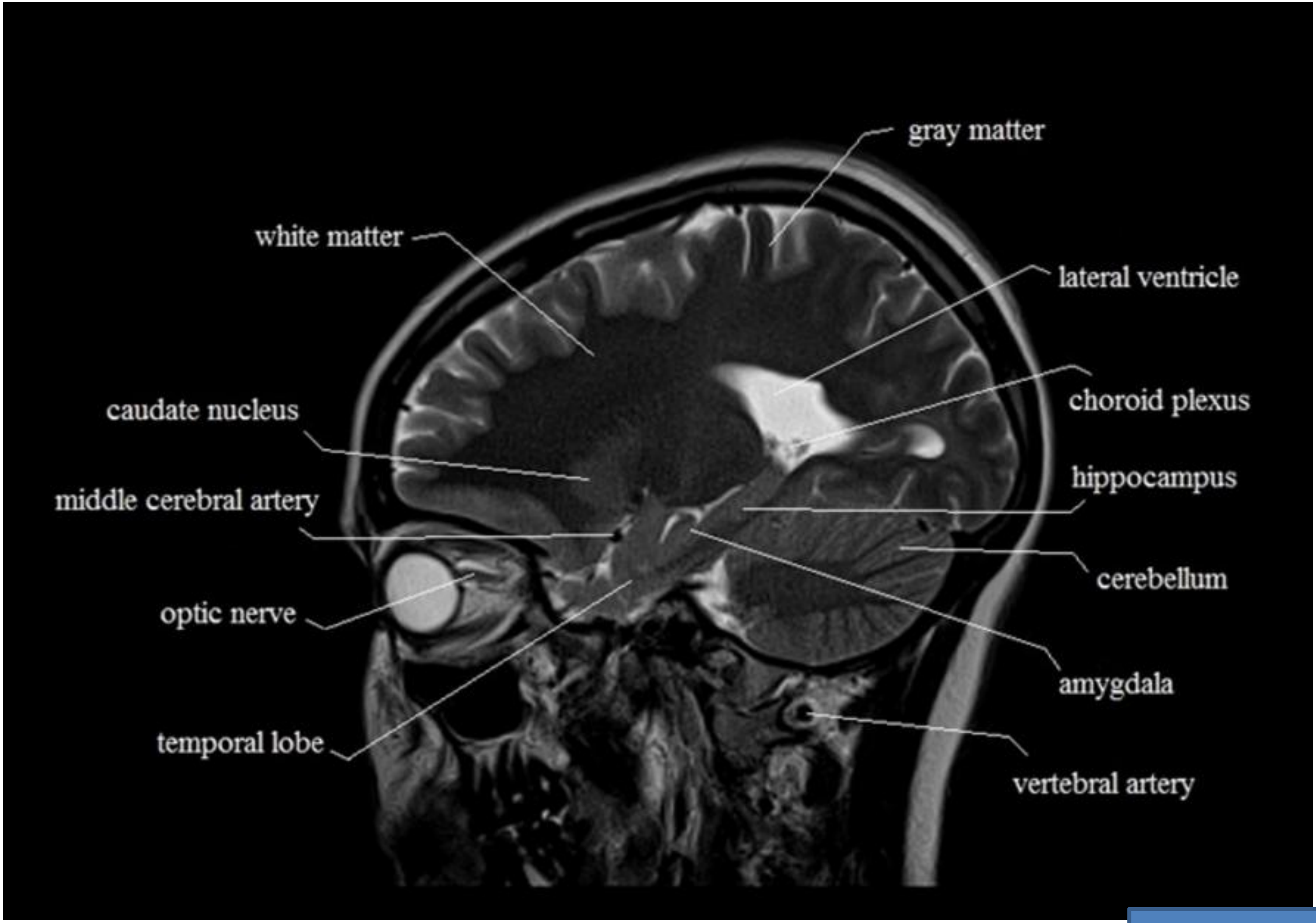

third ventricle
body of corpus callosum
cerebral peduncle of midbrain
lateral ventricle
thalamus
genu of corpus callosum
hypothalamus
mammillary body
optic chiasm
pituitary gland
pons
basilar artery
uvula elevation
medulla oblongata
superior sagittal sinus
internal cerebral vein
splenium of corpus callosum
vein of galen
pineal gland
inferior colliculus of midbrain
straight sinus
superior medullary velum
trochlear herophili
cerebellum
aqueduct of sylvius
fourth ventricle
cerebellar tonsil
cisterna magna
spinal cord

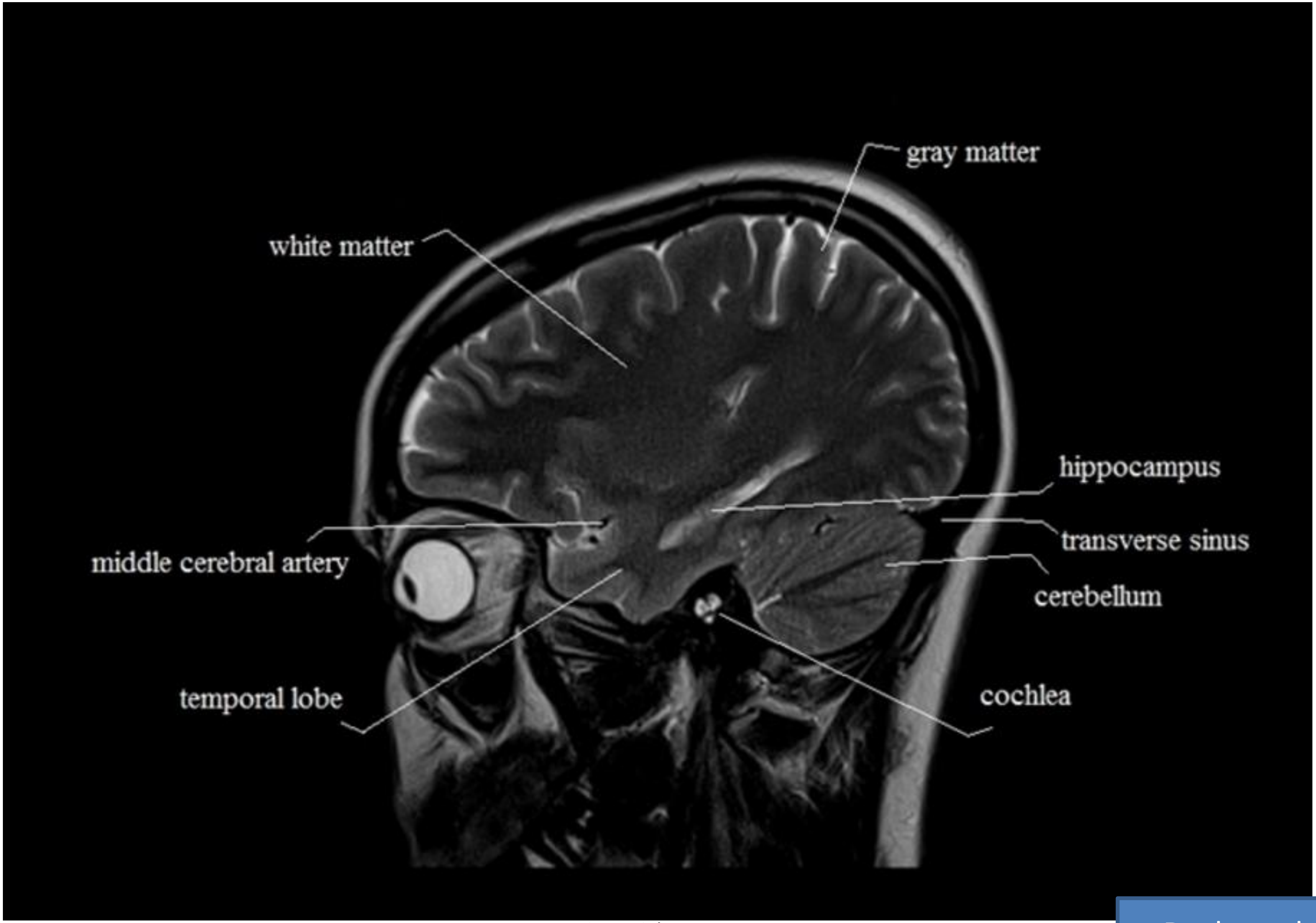

thalamus
body of corpus callosum
gray matter
lateral ventricle
caudate nucleus
splenium of corpus callosum
genu of corpus callosum
hypothalamus
cerebellum
fourth ventricle
pons
vertebral artery

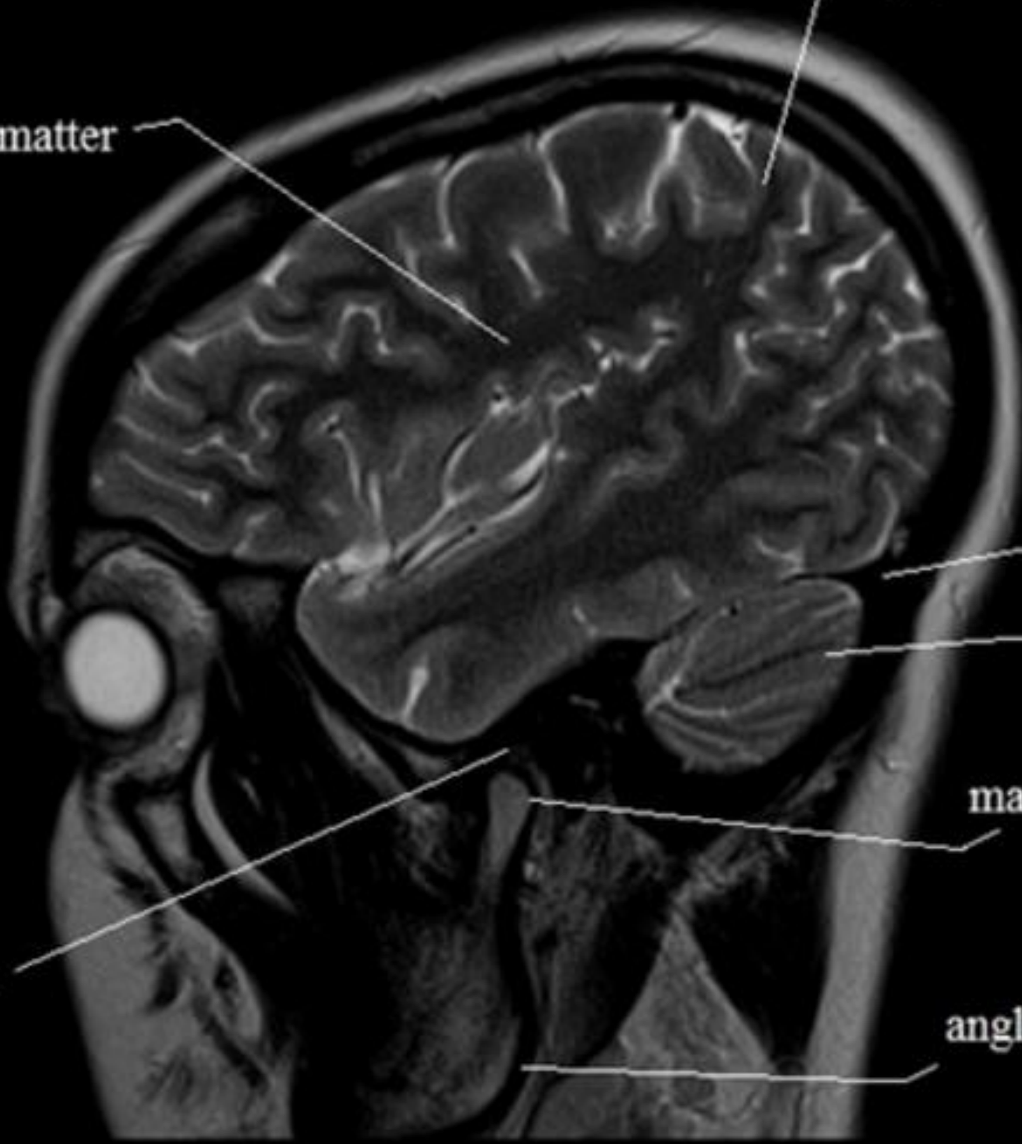

thalamus
gray matter
lateral ventricle
caudate nucleus
transverse sinus
cerebellum
internal carotid artery
vertebral artery

Dr.shamol


putamen
gray matter
lateral ventricle
caudate nucleus
posterior cerebral artery
transverse sinus
middle cerebral artery
cerebellum
vertebral artery


gray matter
white matter
lateral ventricle
caudate nucleus
choroid plexus
middle cerebral artery
hippocampus
cerebellum
optic nerve
amygdala
temporal lobe
vertebral artery


gray matter
white matter
hippocampus
transverse sinus
middle cerebral artery
cerebellum
temporal lobe
cochlea


gray matter
white matter
transverse sinus
cerebellum
mandibular condyle
temporomandibular joint
angle of jaw

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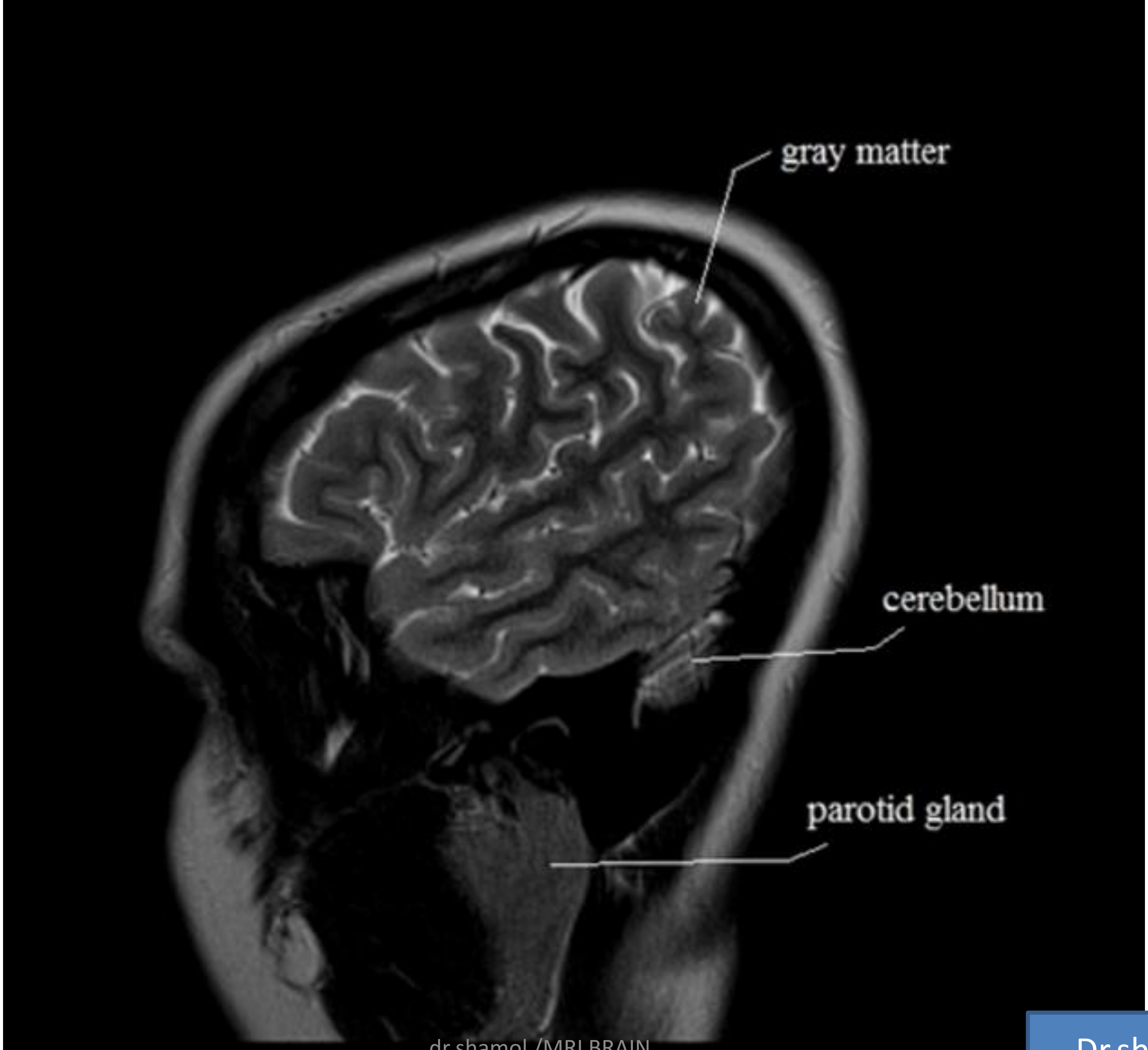

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NOW IDENTIFY THE STRUCTURE'S BY YOUR SELF

ROW -1

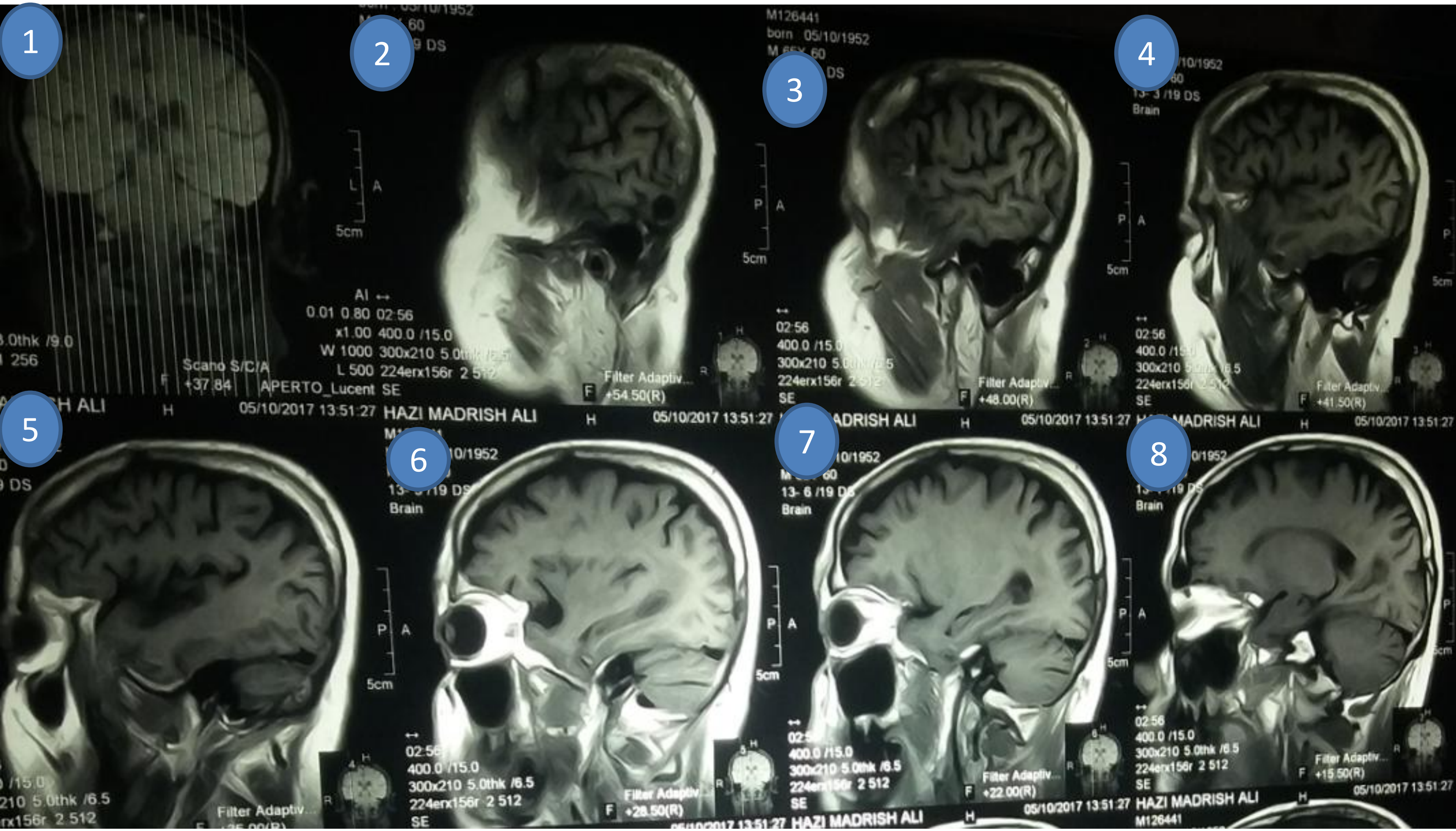
ROW -2

ROW -3

ROW -4

ROW -5

dr shamol /MRI BRAIN


1
2
3
4
5
6
7
8
HAZI MADRISH ALI
05/10/2017 13:51:27
born 05/10/1952
M126441
Brain
5cm
Filter Adaptiv...
+54.50(R)
+48.00(R)
+41.50(R)
+28.50(R)
+22.00(R)
+15.50(R)
02:56
400.0 /15.0
300x210 5.0thk /6.5
224erx156r 2 512
SE
APERTO_Lucent


9
10
11
12
13
!4
15
16
17
18
19
20

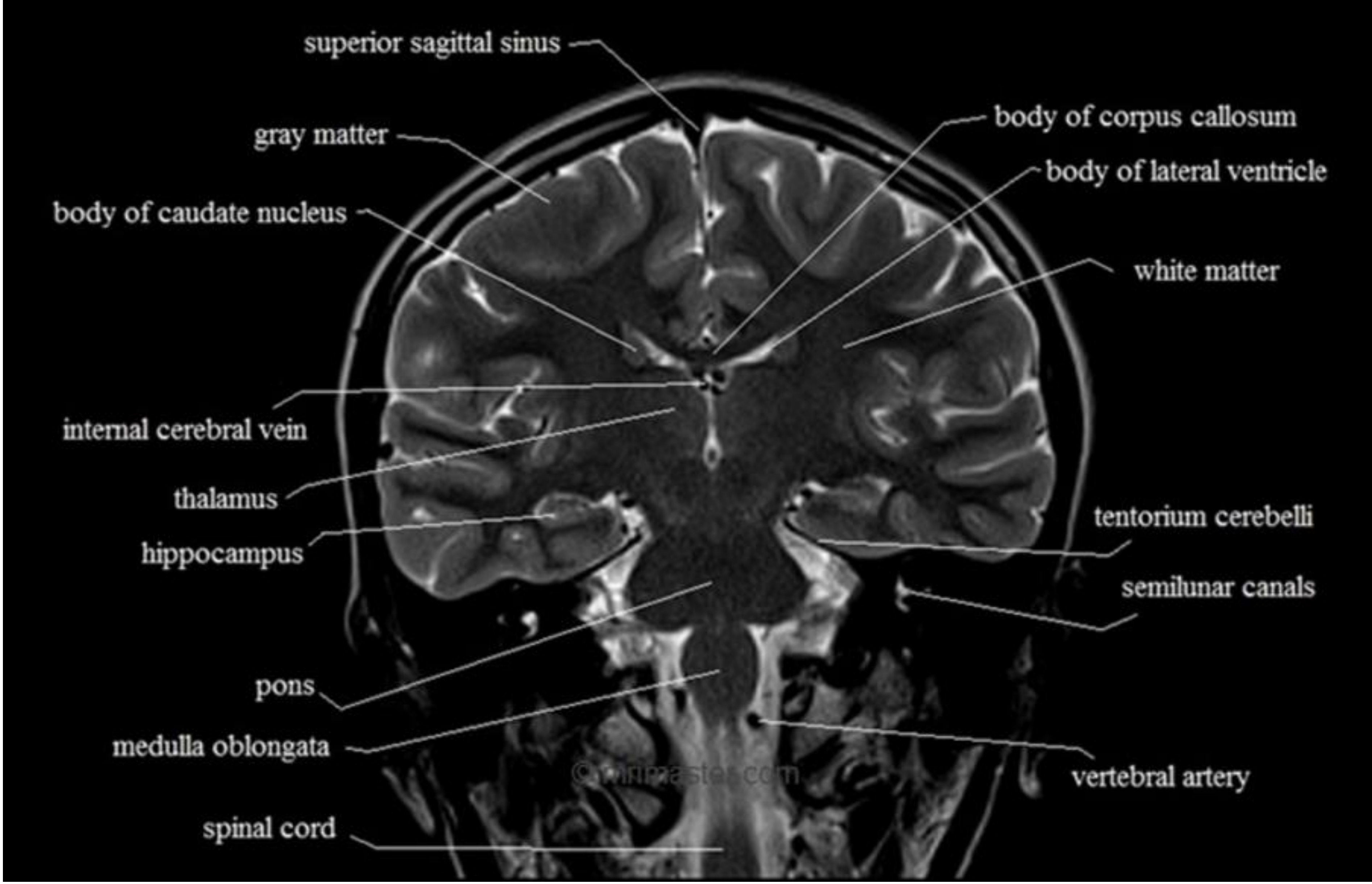
CORONAL

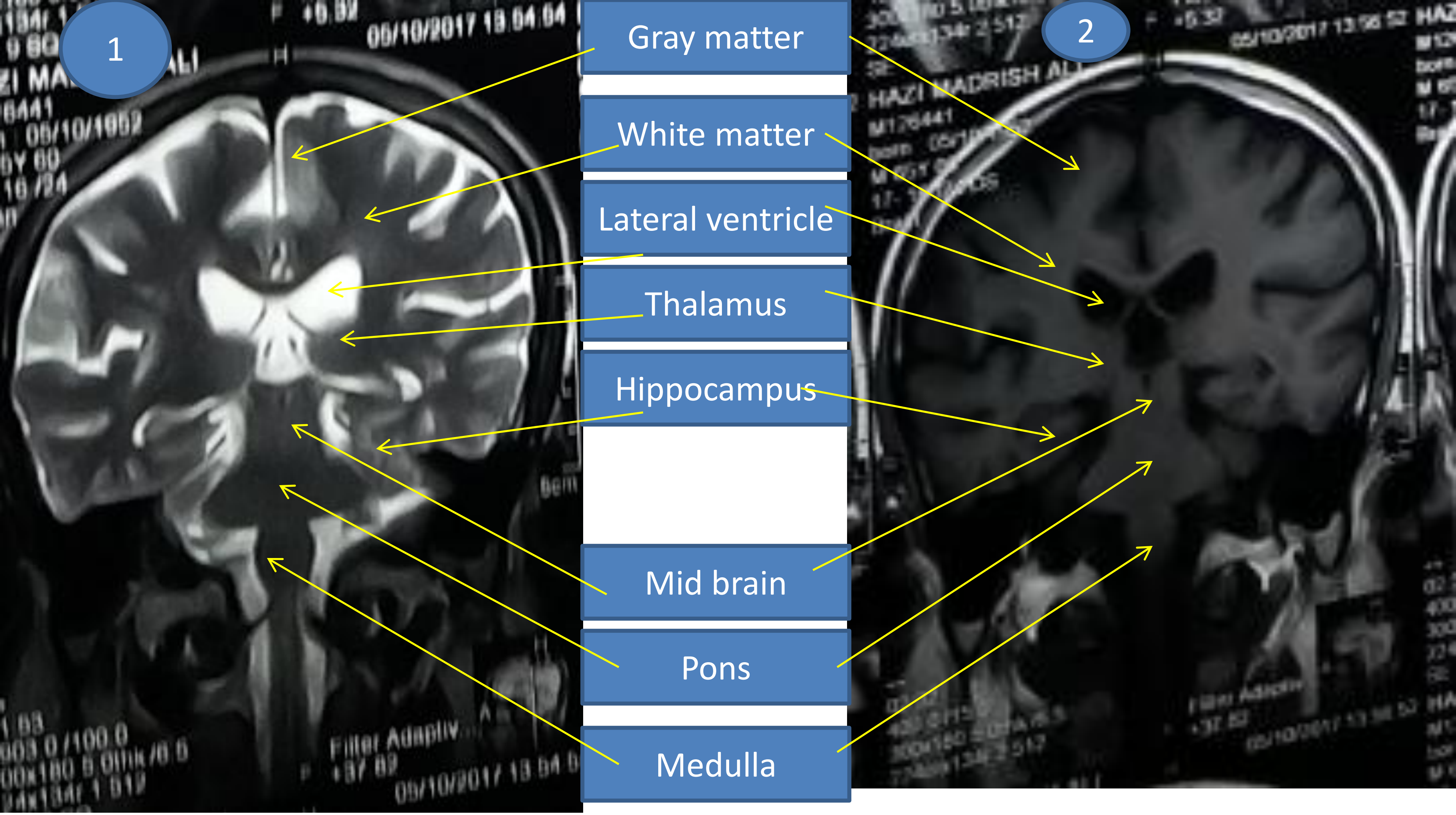

9300
rn : 07/22/1960
57Y 60
9- 14 /24
rain
R
05:19
4903.0 /100.0
FSE 7 SQ
H
ABUL HASHEM
679300
born 07/22/1960
M 57Y 60
19- 15 /24
Brain
5cm
R
05 19
4903 0 /100 0
FC
Filter Adaptiv
+27 92
F

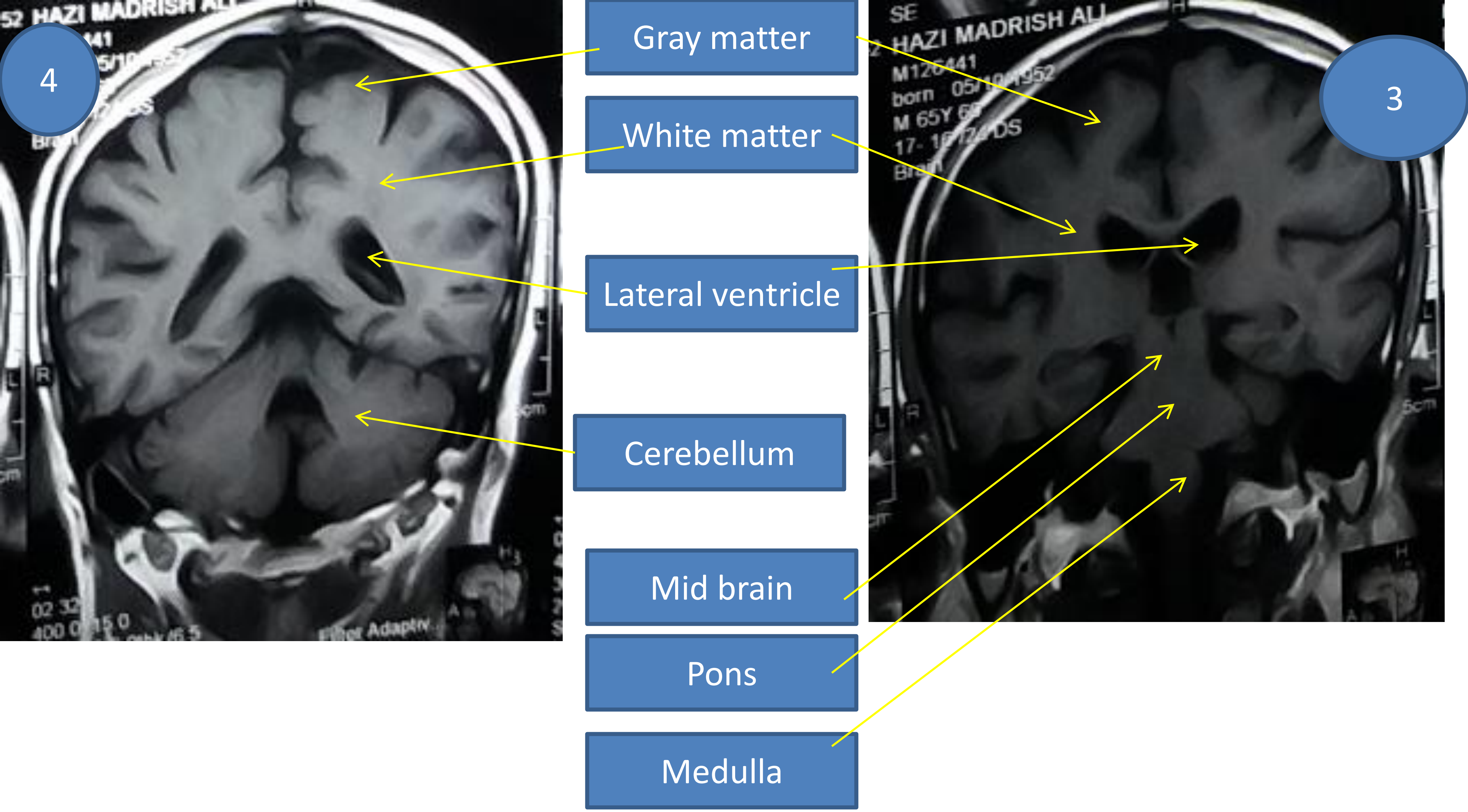

T1


T2

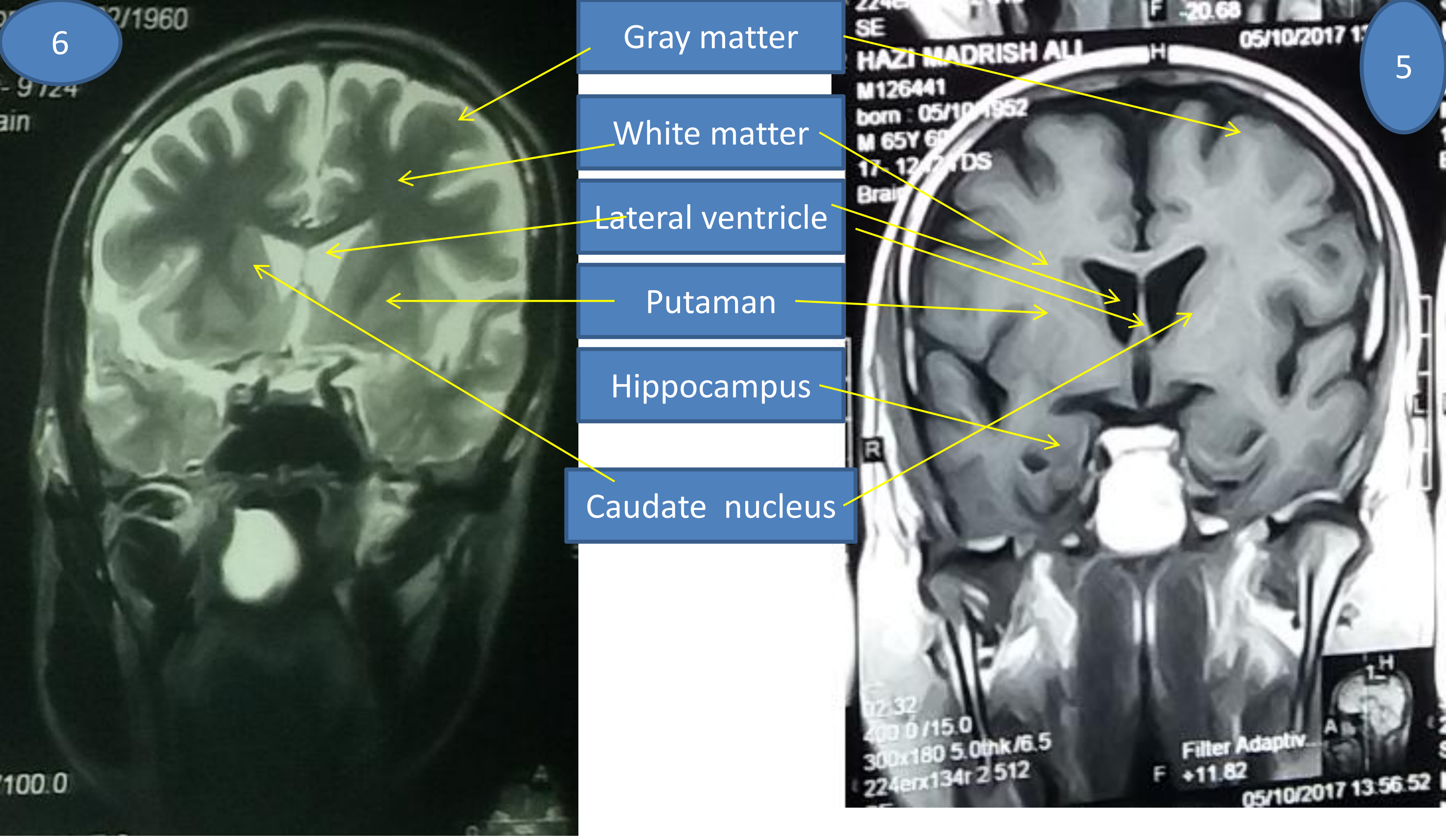
MOST IMPORTANT STRUCTURE OF CRONAL FILMS
YOU HAVE TO KNOW

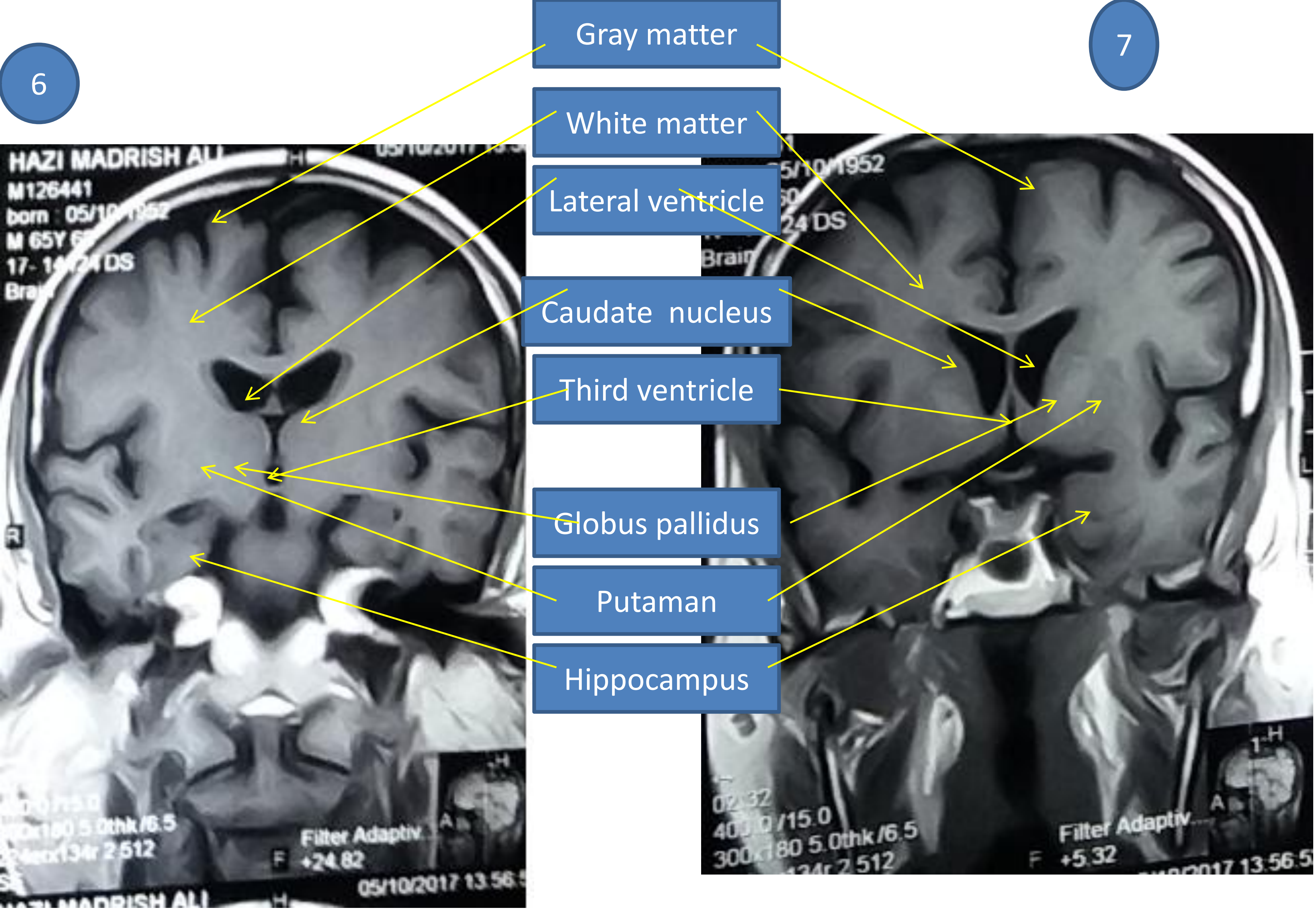

superior sagittal sinus
gray matter
body of caudate nucleus
internal cerebral vein
thalamus
hippocampus
pons
medulla oblongata
spinal cord
body of corpus callosum
body of lateral ventricle
white matter
tentorium cerebelli
semilunar canals
vertebral artery


1
2
Gray matter
White matter
Lateral ventricle
Thalamus
Hippocampus
Mid brain
Pons
Medulla


4
3
Gray matter
White matter
Lateral ventricle
Cerebellum
Mid brain
Pons
Medulla

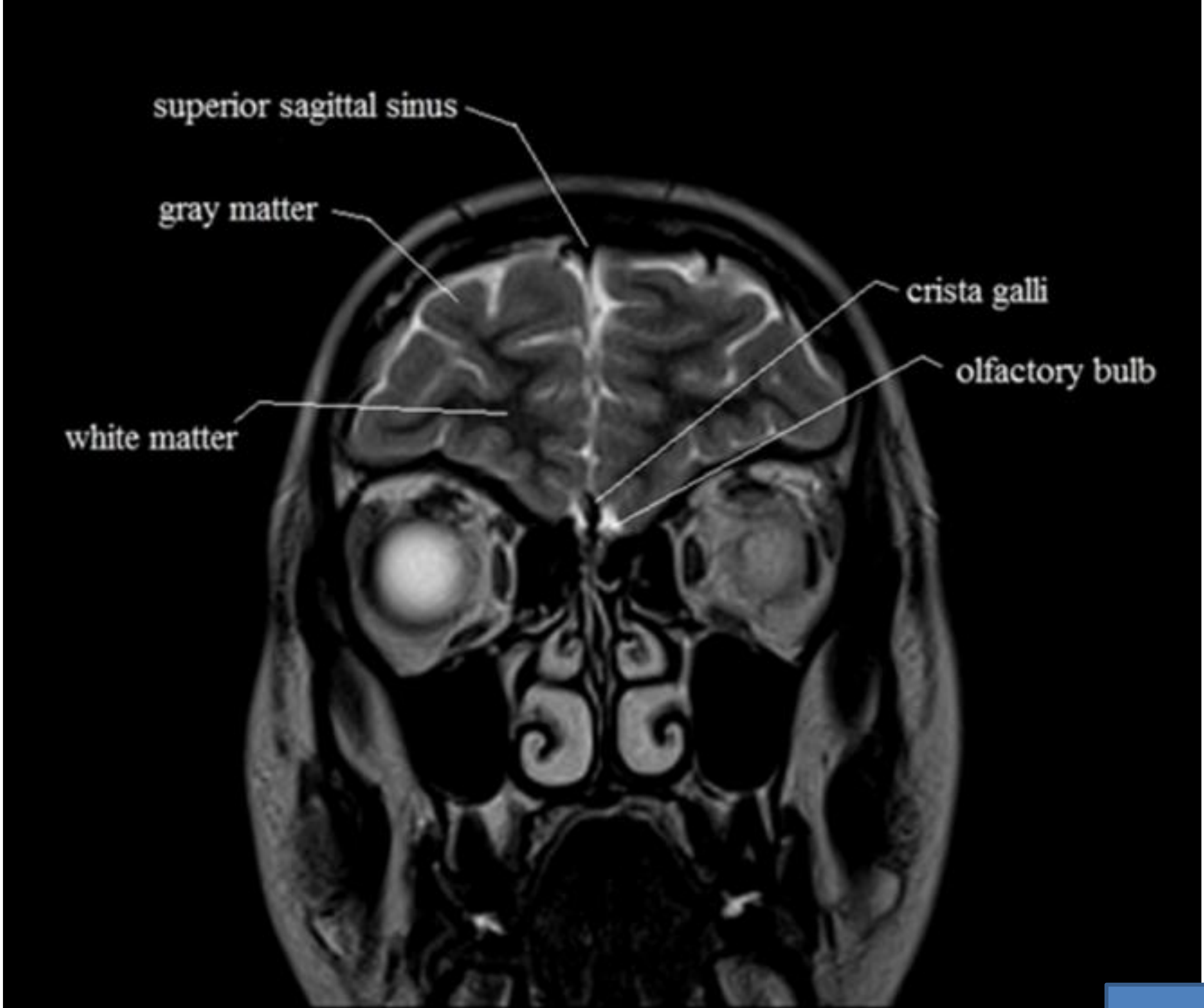
Dr.shamol

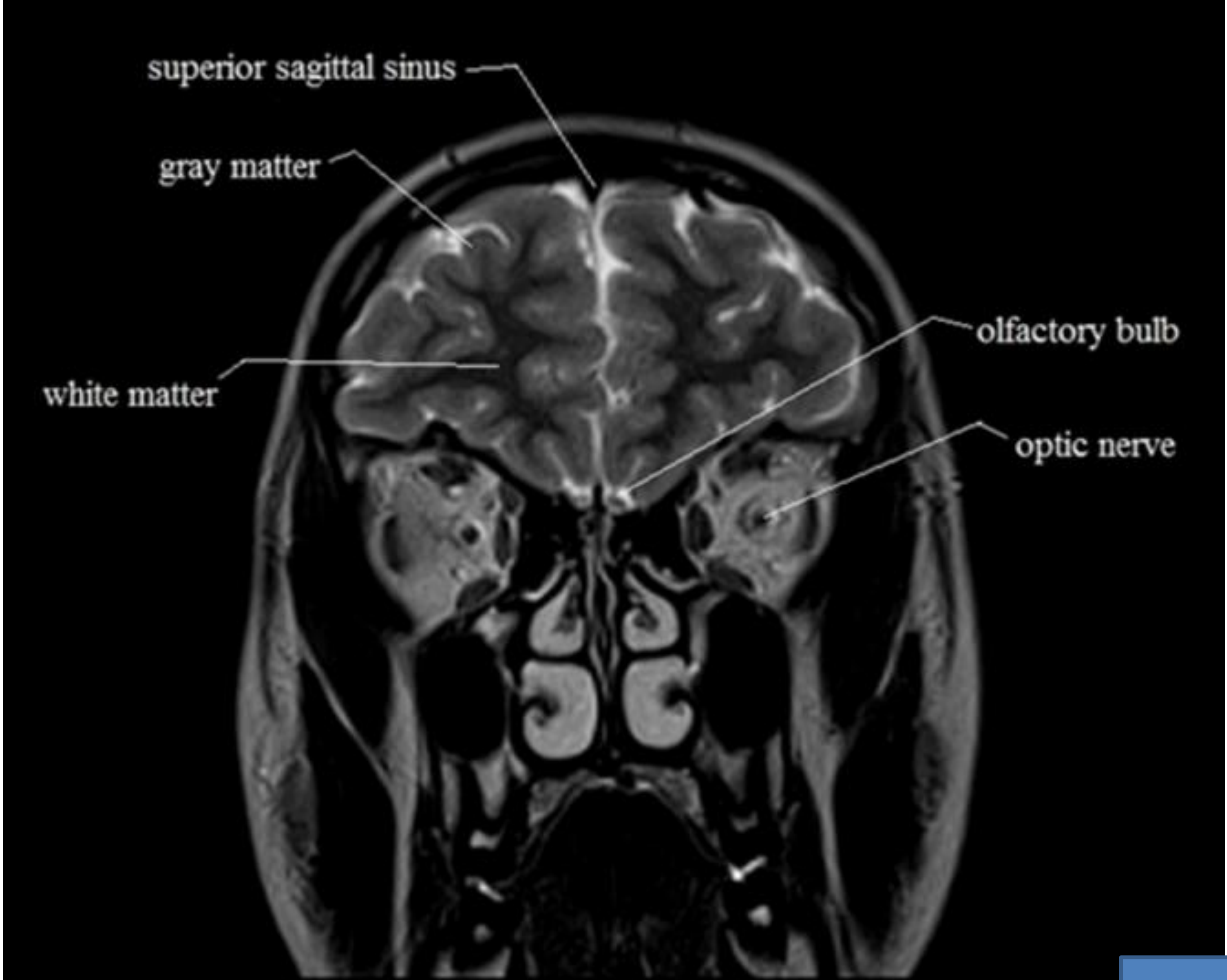

6
5
Gray matter
White matter
Lateral ventricle
Putaman
Hippocampus
Caudate nucleus

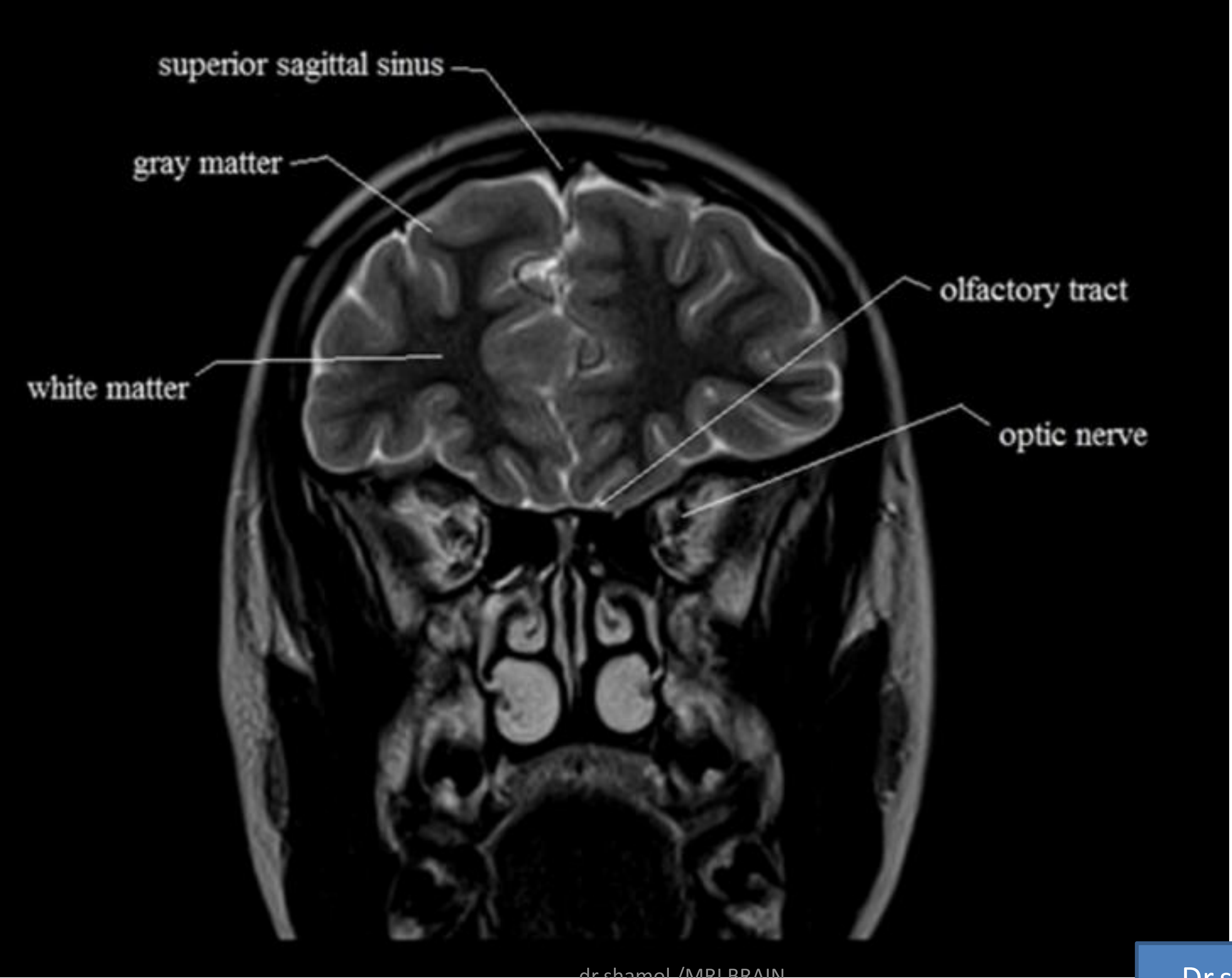

6
7
Gray matter
White matter
Lateral ventricle
Caudate nucleus
Third ventricle
Globus pallidus
Putaman
Hippocampus
HAZI MADRISH ALI
M126441
Filter Adaptiv.
+24.82
Filter Adaptiv...
+5.32

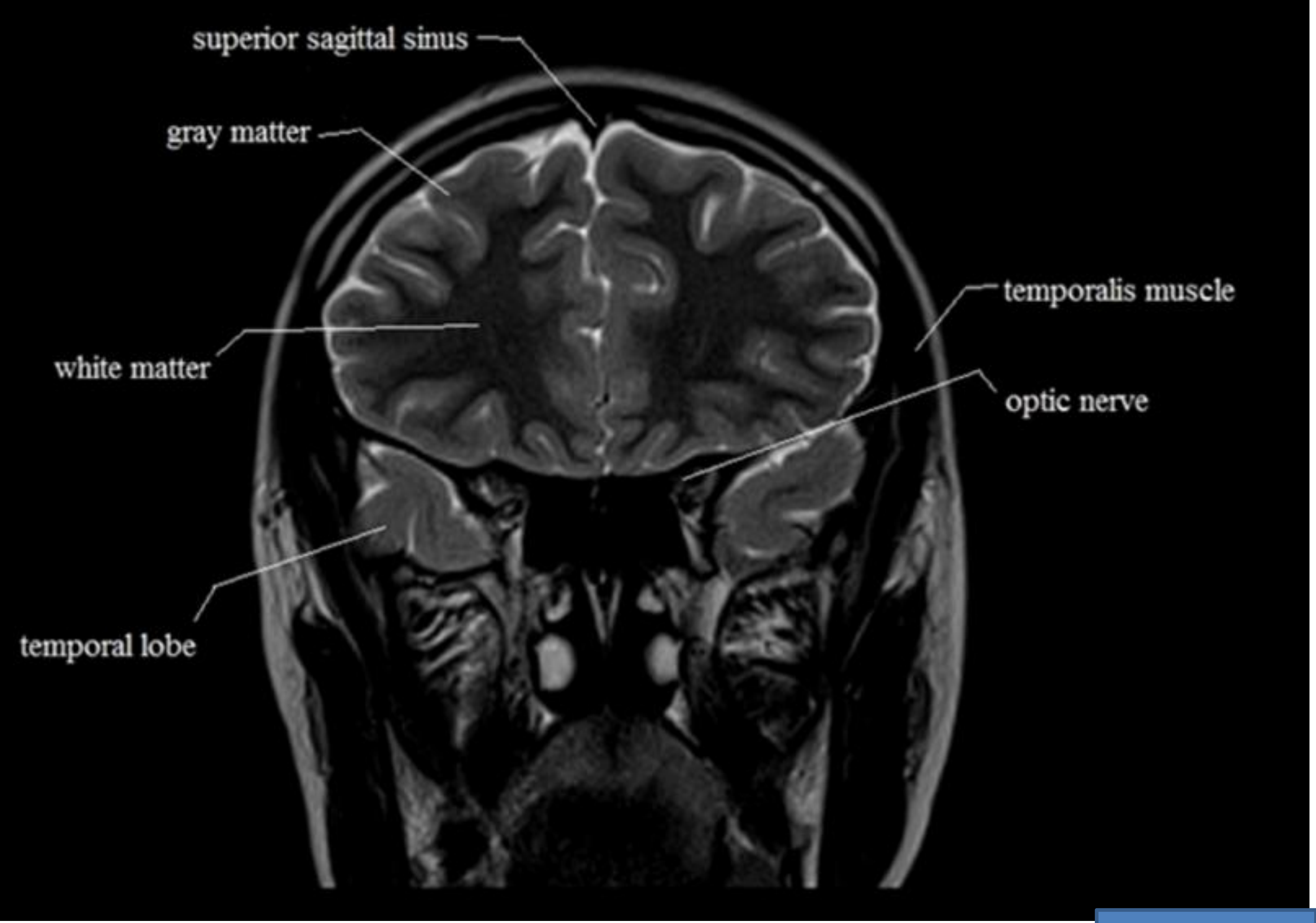
CORONAL
Structure A to Z

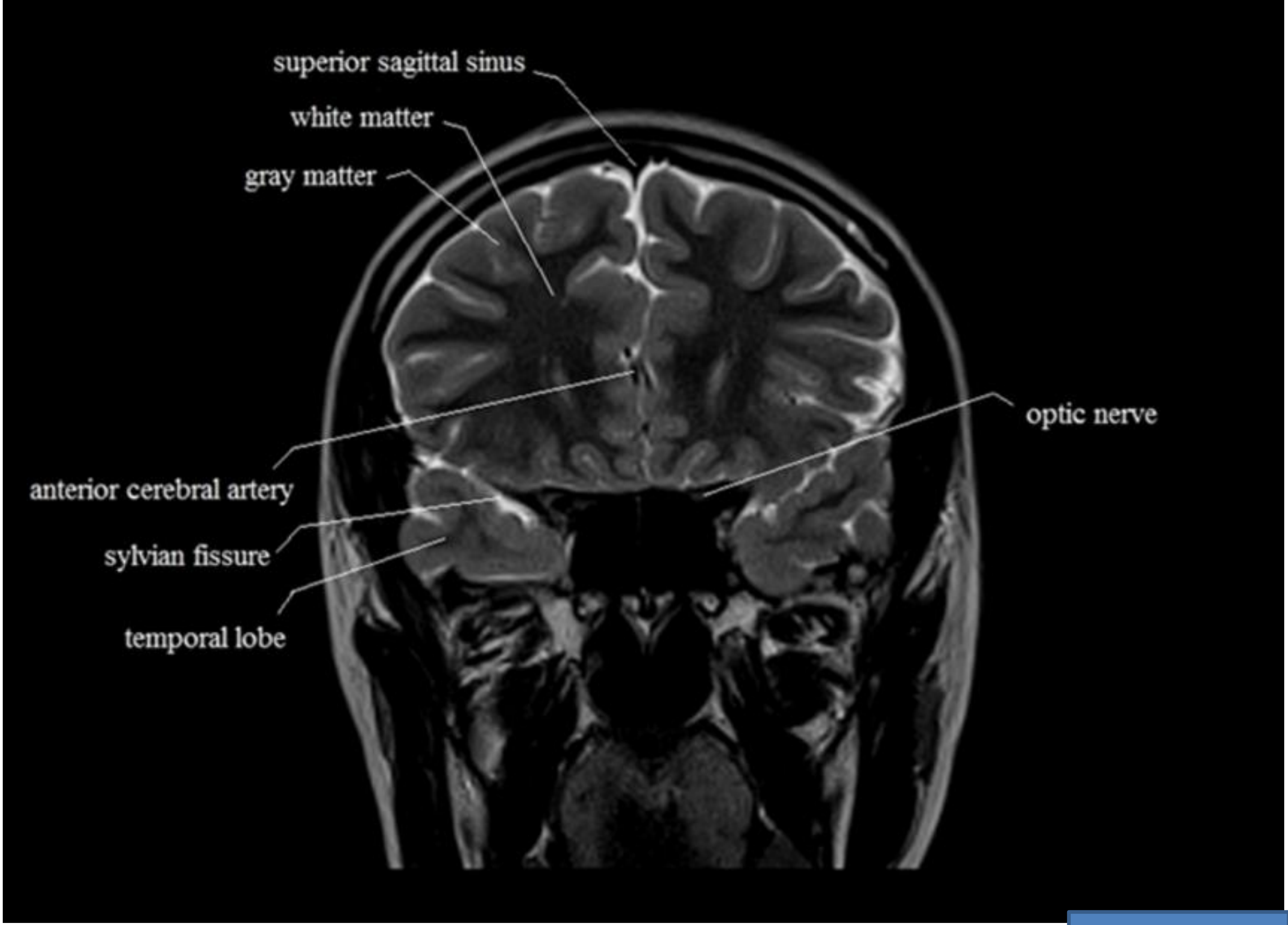
Sequence
FROM FRONT → BACK


superior sagittal sinus
gray matter
crista galli
olfactory bulb
white matter

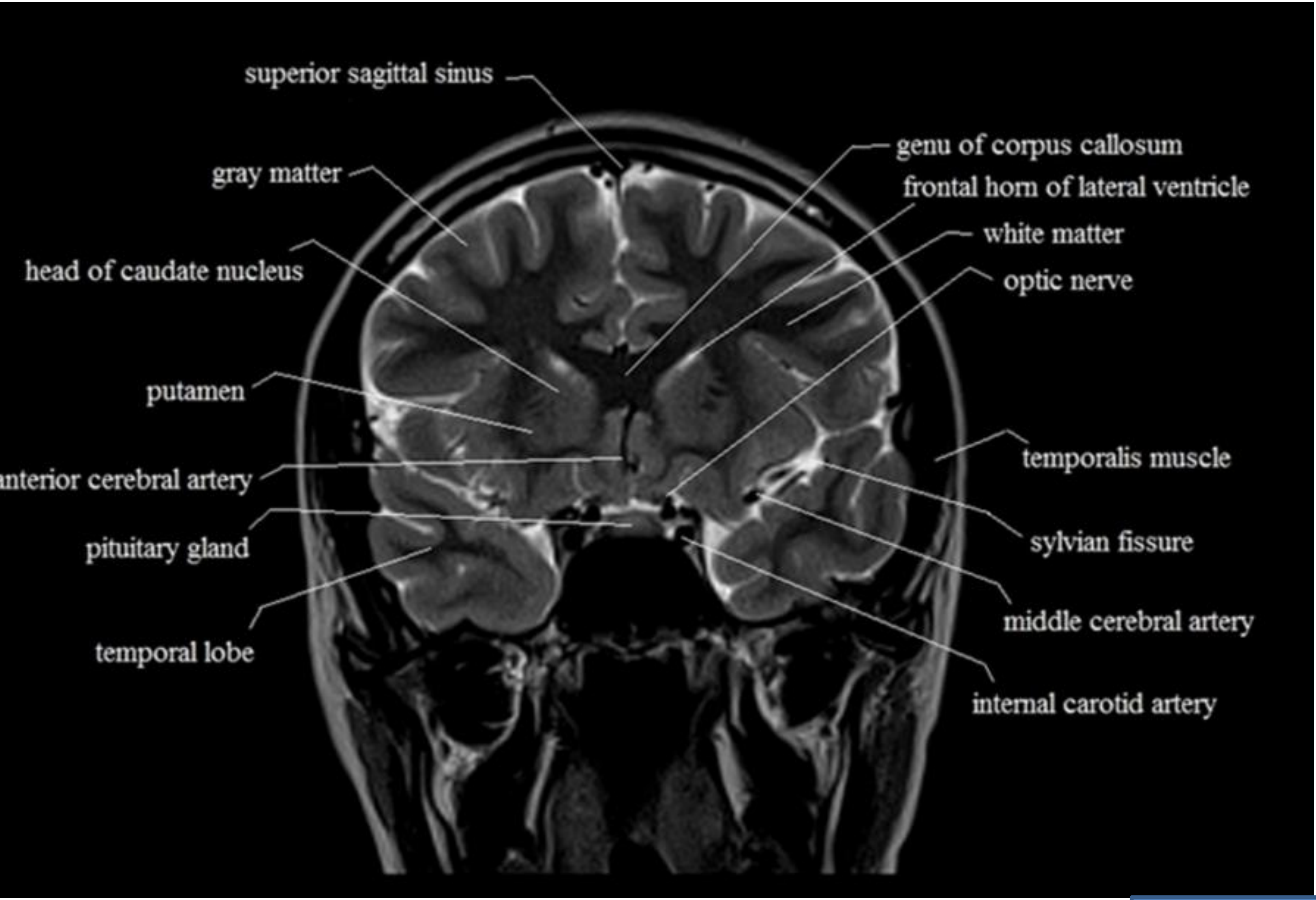

superior sagittal sinus
gray matter
white matter
olfactory bulb
optic nerve

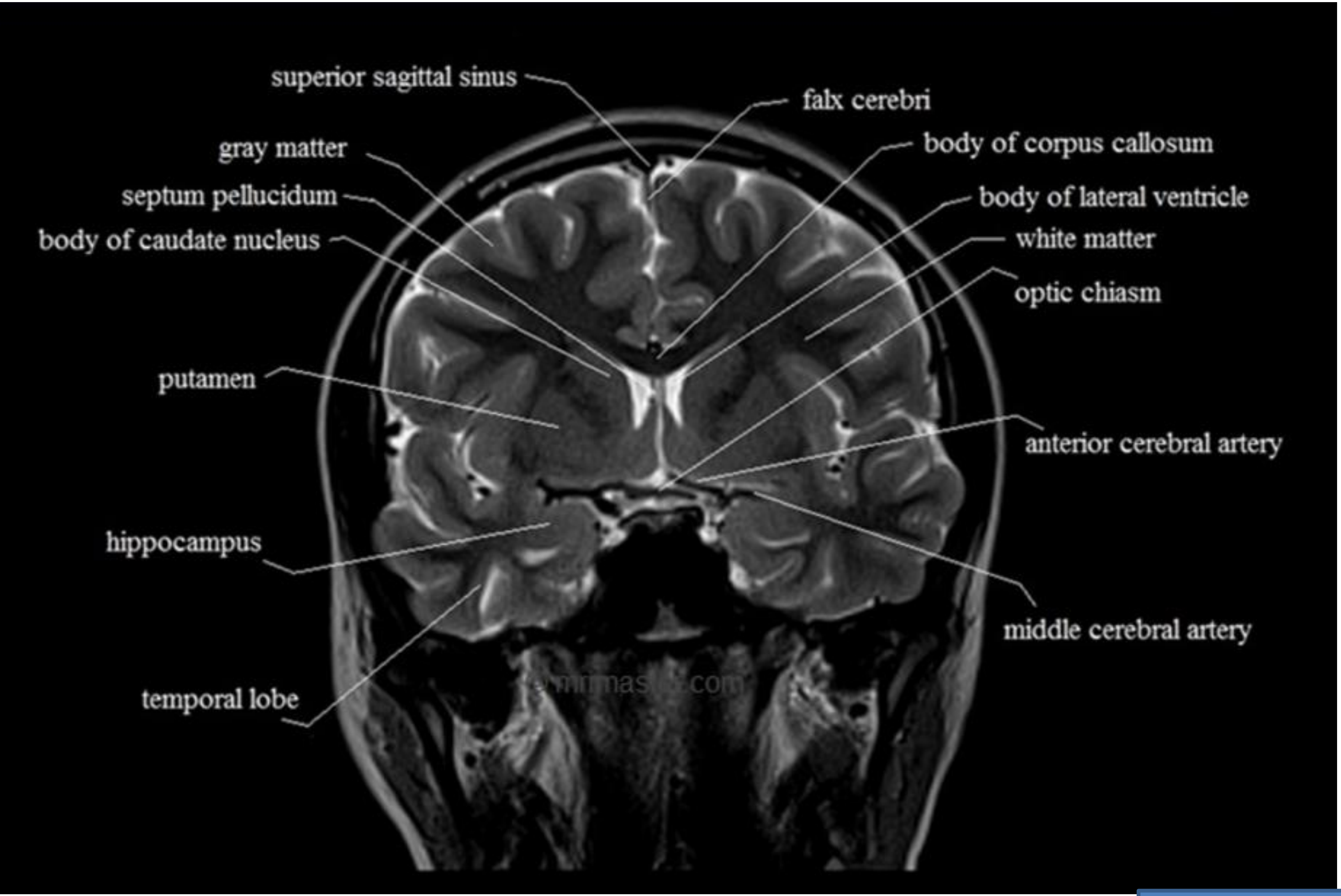

superior sagittal sinus
gray matter
white matter
olfactory tract
optic nerve

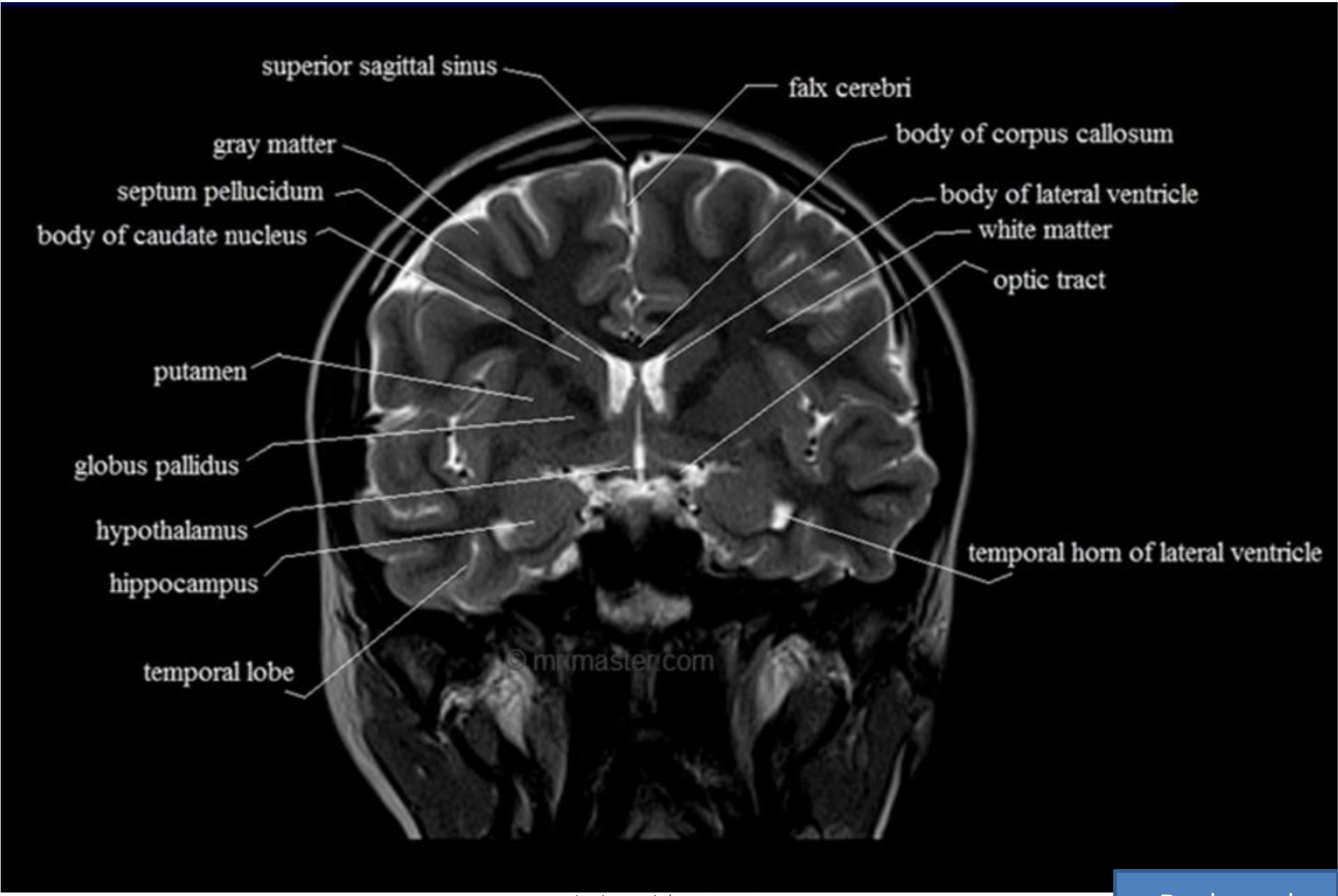

superior sagittal sinus
gray matter
temporalis muscle
white matter
optic nerve
temporal lobe

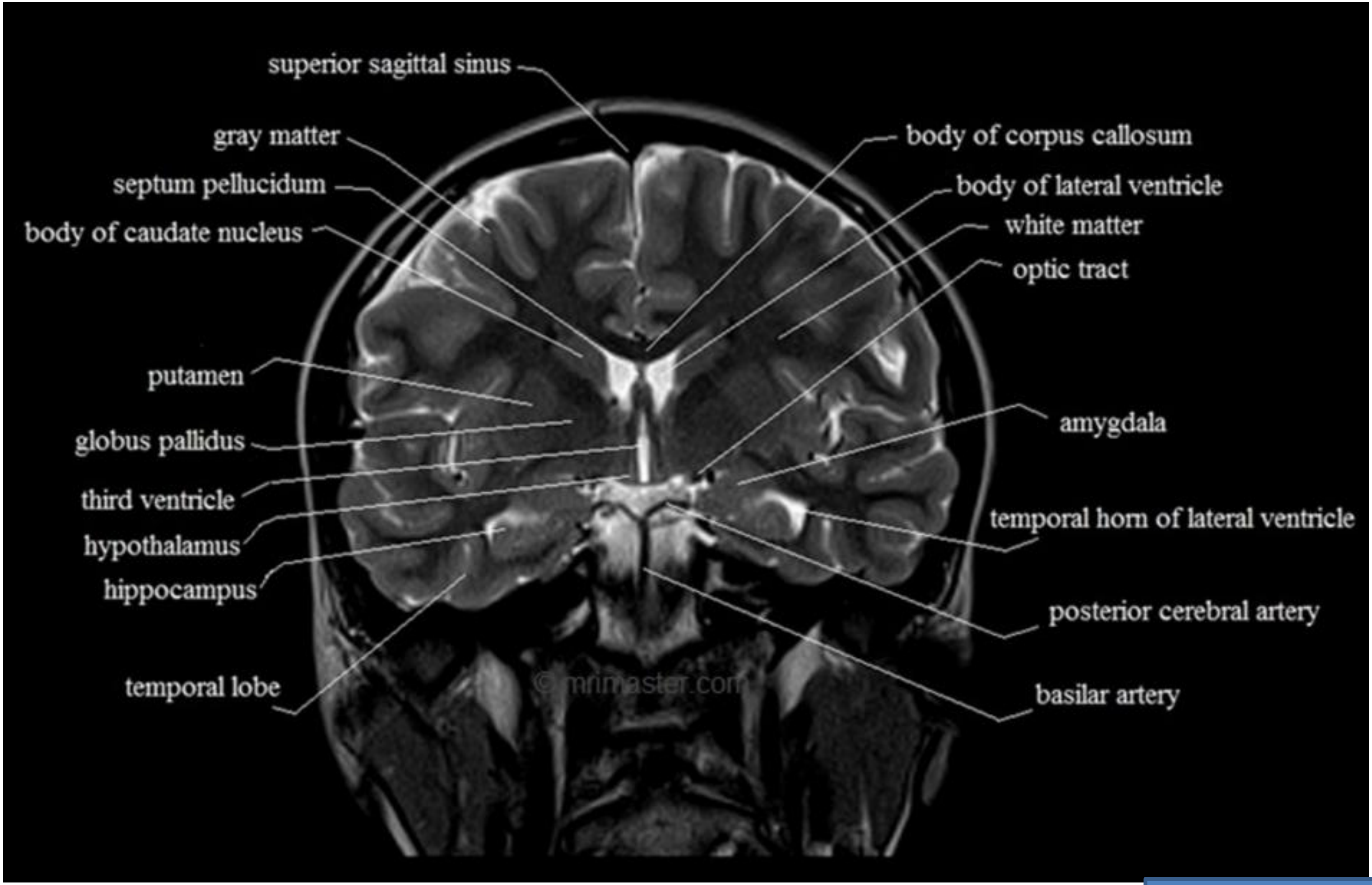

superior sagittal sinus
white matter
gray matter
optic nerve
anterior cerebral artery
sylvian fissure
temporal lobe

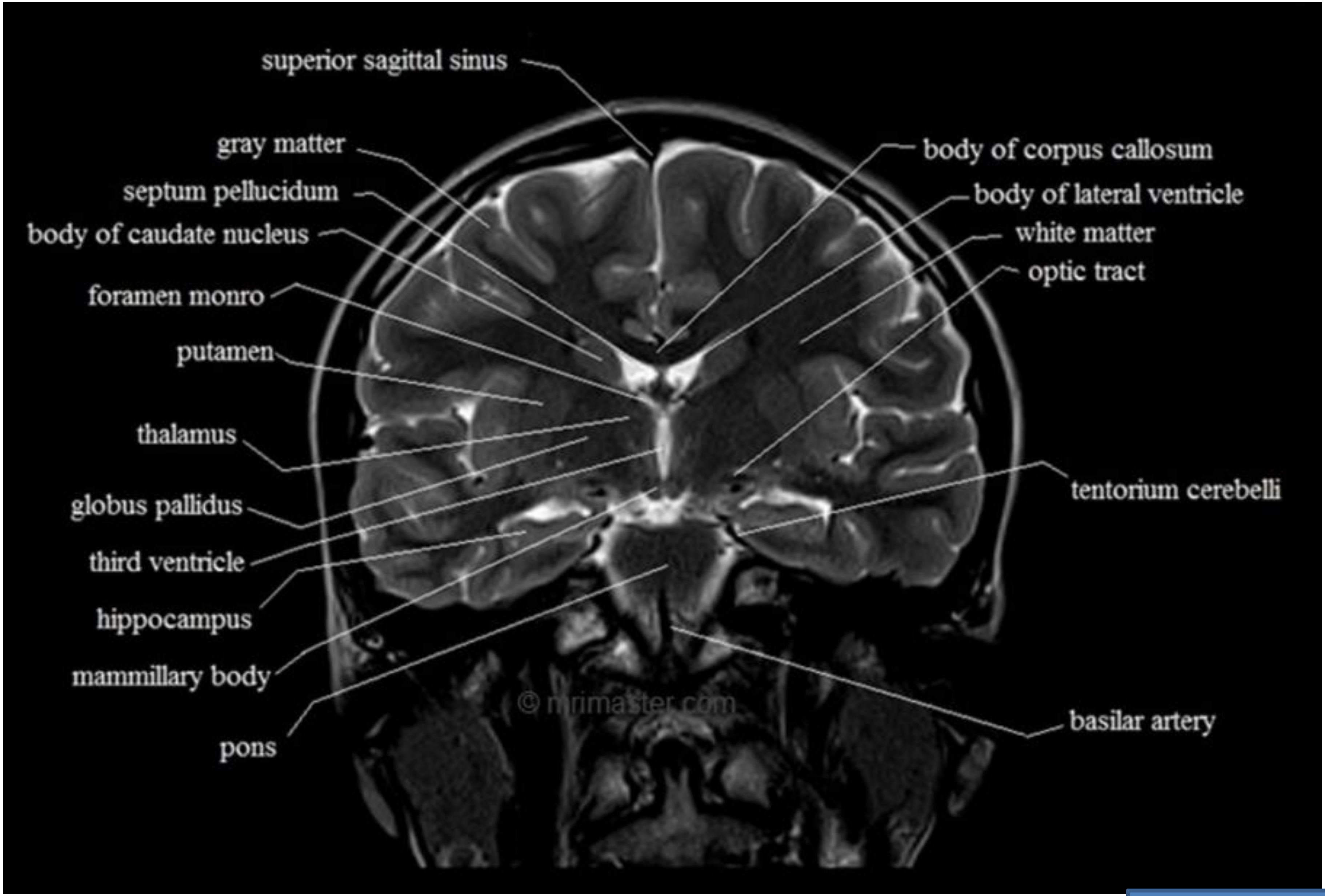
Dr.shamol

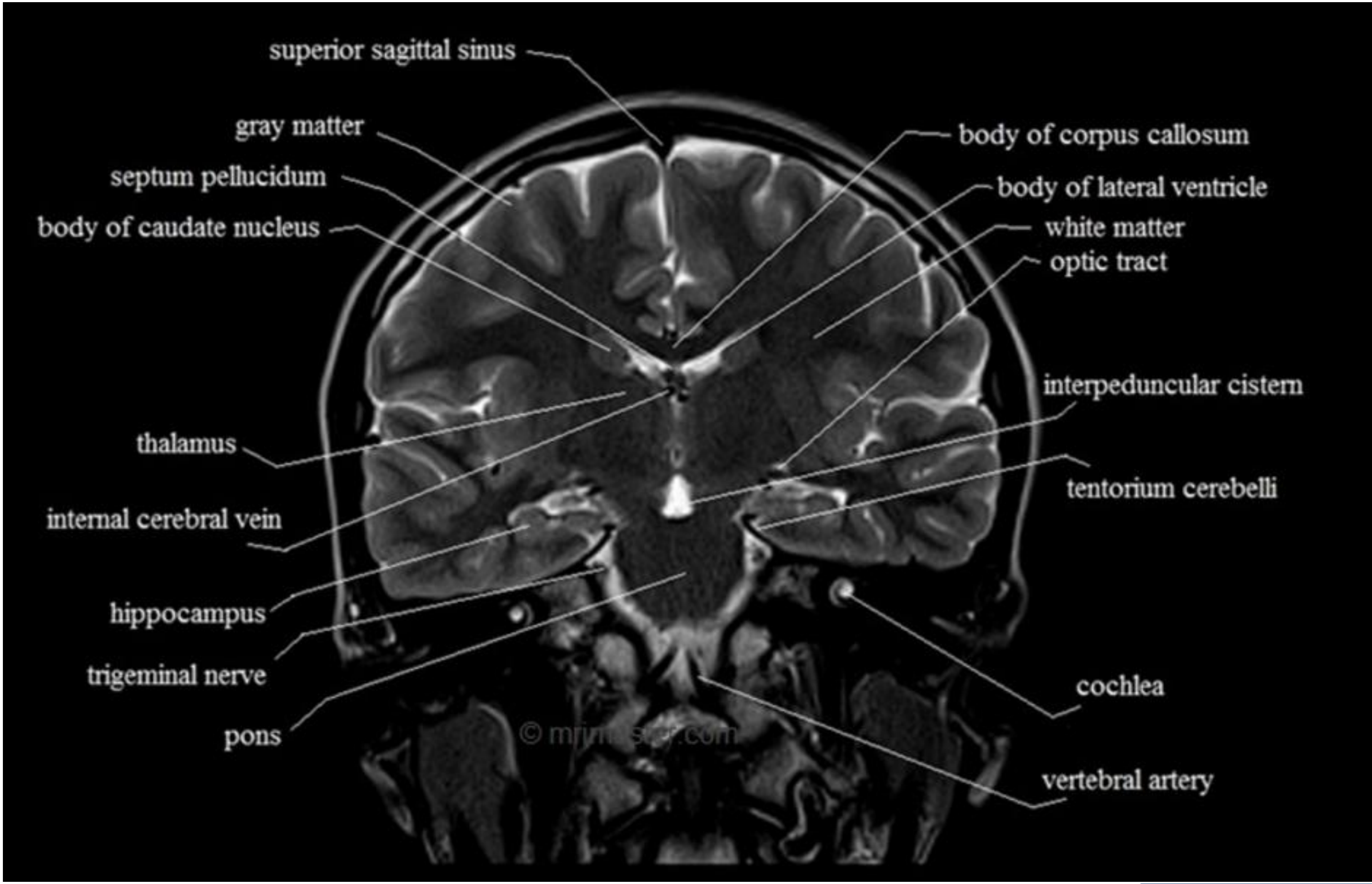

superior sagittal sinus
gray matter
head of caudate nucleus
putamen
nterior cerebral artery
pituitary gland
temporal lobe
genu of corpus callosum
frontal horn of lateral ventricle
white matter
optic nerve
temporalis muscle
sylvian fissure
middle cerebral artery
internal carotid artery


superior sagittal sinus
falx cerebri
gray matter
body of corpus callosum
septum pellucidum
body of lateral ventricle
body of caudate nucleus
white matter
optic chiasm
putamen
anterior cerebral artery
hippocampus
middle cerebral artery
temporal lobe


superior sagittal sinus
falx cerebri
gray matter
body of corpus callosum
septum pellucidum
body of lateral ventricle
body of caudate nucleus
white matter
optic tract
putamen
globus pallidus
hypothalamus
hippocampus
temporal horn of lateral ventricle
temporal lobe
© mrimaster.com


superior sagittal sinus
gray matter
septum pellucidum
body of caudate nucleus
putamen
globus pallidus
third ventricle
hypothalamus
hippocampus
temporal lobe
body of corpus callosum
body of lateral ventricle
white matter
optic tract
amygdala
temporal horn of lateral ventricle
posterior cerebral artery
basilar artery
© mrimaster.com


superior sagittal sinus
gray matter
septum pellucidum
body of caudate nucleus
foramen monro
putamen
thalamus
globus pallidus
third ventricle
hippocampus
mammillary body
pons
body of corpus callosum
body of lateral ventricle
white matter
optic tract
tentorium cerebelli
basilar artery
© mrimaster.com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gray matter
septum pellucidum
body of caudate nucleus
thalamus
internal cerebral vein
hippocampus
trigeminal nerve
pons
body of corpus callosum
body of lateral ventricle
white matter
optic tract
interpeduncular cistern
tentorium cerebelli
cochlea
vertebral artery
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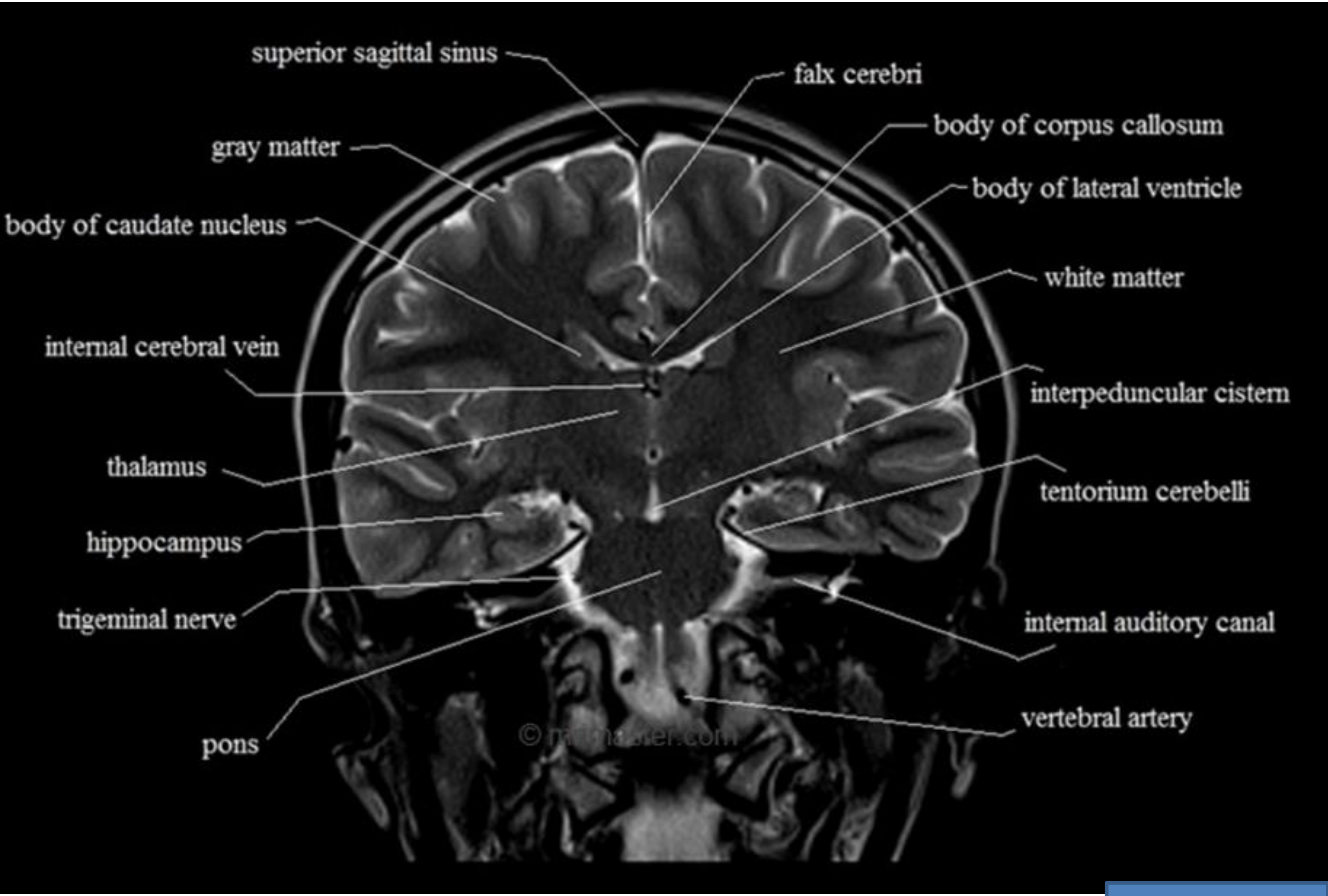
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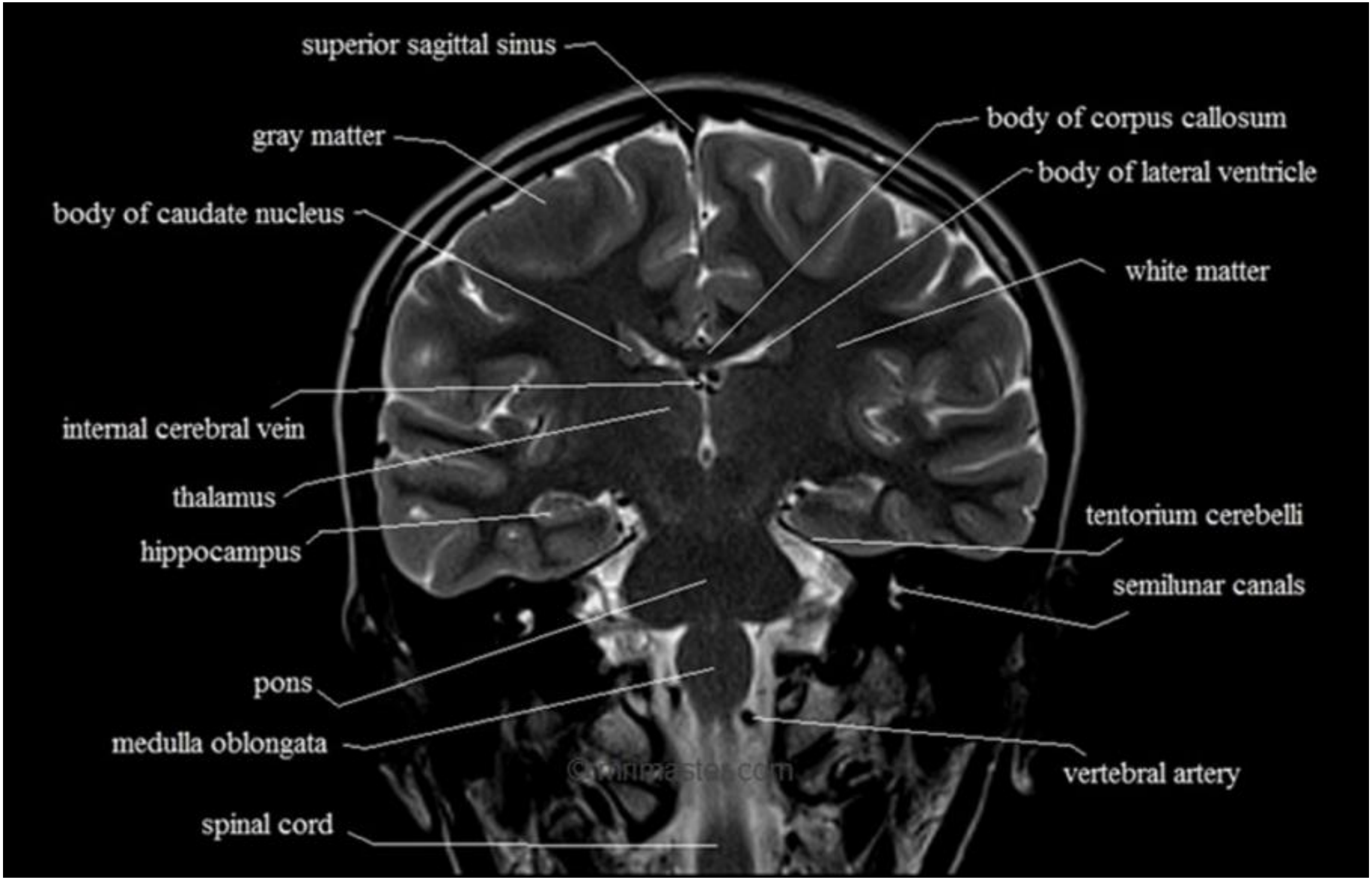
falx cerebri
body of corpus callosum
gray matter
body of lateral ventricle
body of caudate nucleus
white matter
internal cerebral vein
interpeduncular cistern
thalamus
tentorium cerebelli
hippocampus
trigeminal nerve
internal auditory canal
vertebral artery
pons
© mrimaster.com


superior sagittal sinus
gray matter
body of caudate nucleus
body of corpus callosum
body of lateral ventricle
white matter
internal cerebral vein
thalamus
hippocampus
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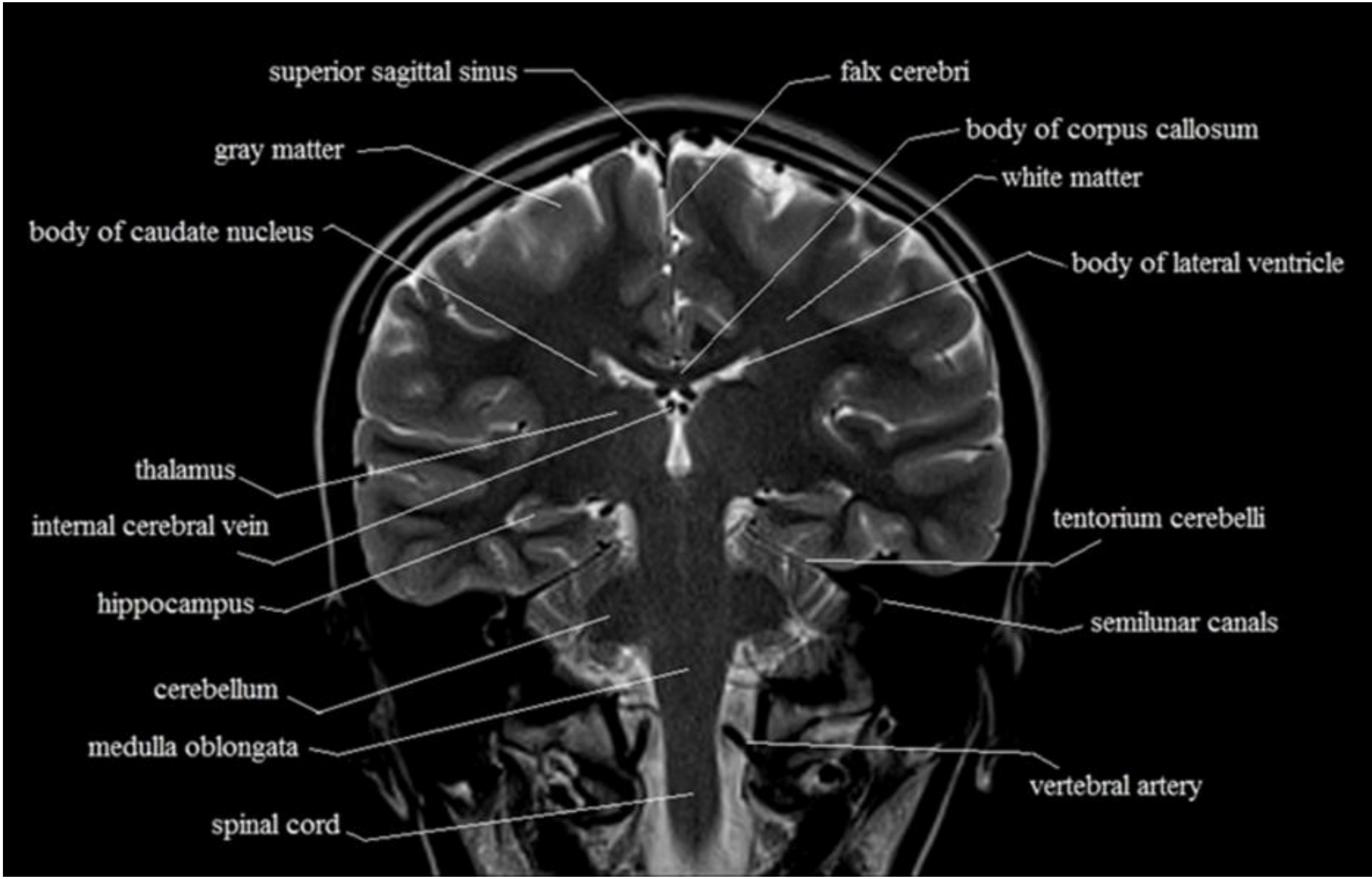
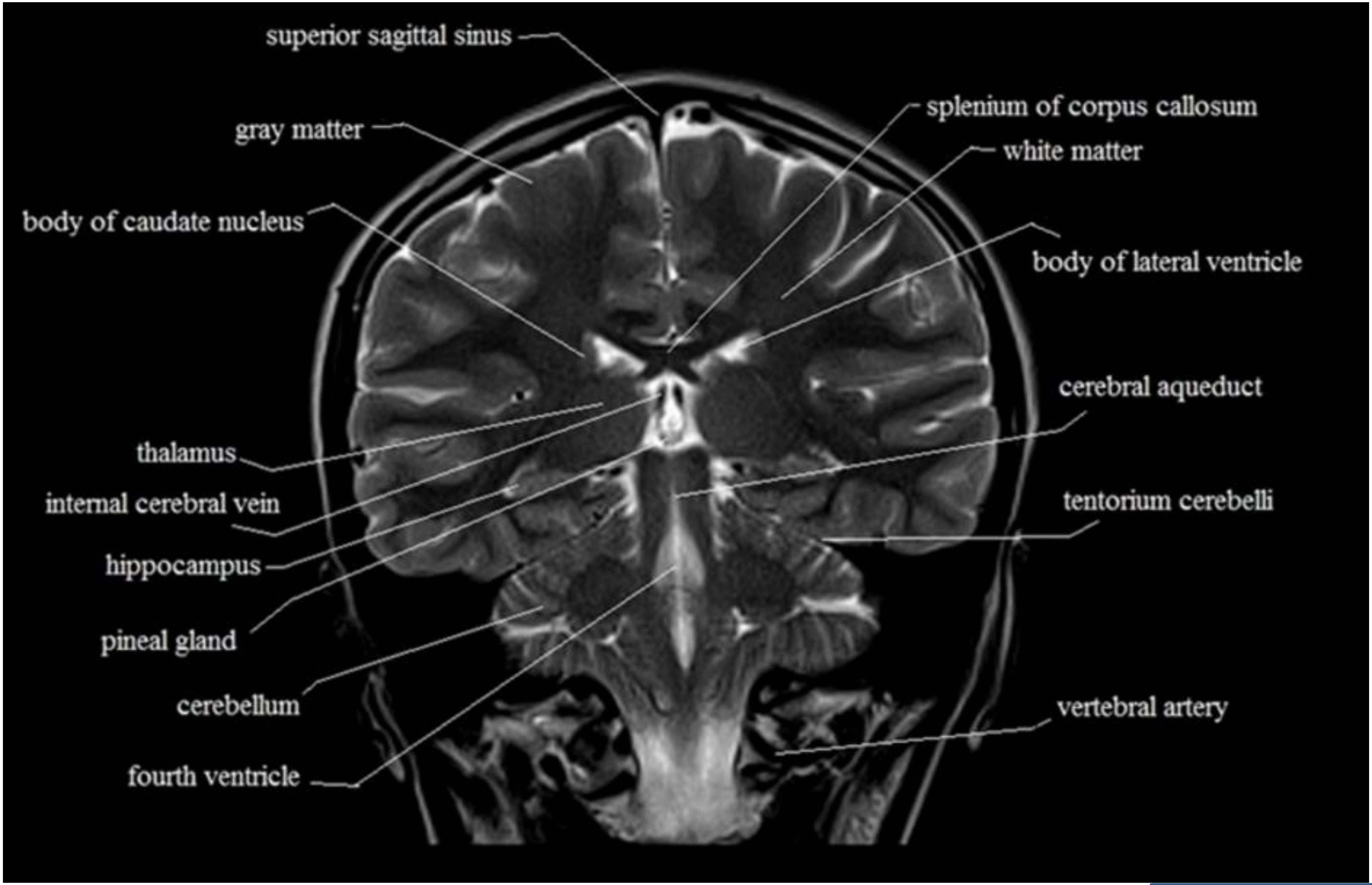
semilunar canals
pons
medulla oblongata
vertebral artery
spinal cord


superior sagittal sinus
falx cerebri
body of corpus callosum
gray matter
white matter
body of caudate nucleus
body of lateral ventricle
thalamus
internal cerebral vein
tentorium cerebelli
hippocampus
semilunar canals
cerebellum
medulla oblongata
vertebral artery
spinal cord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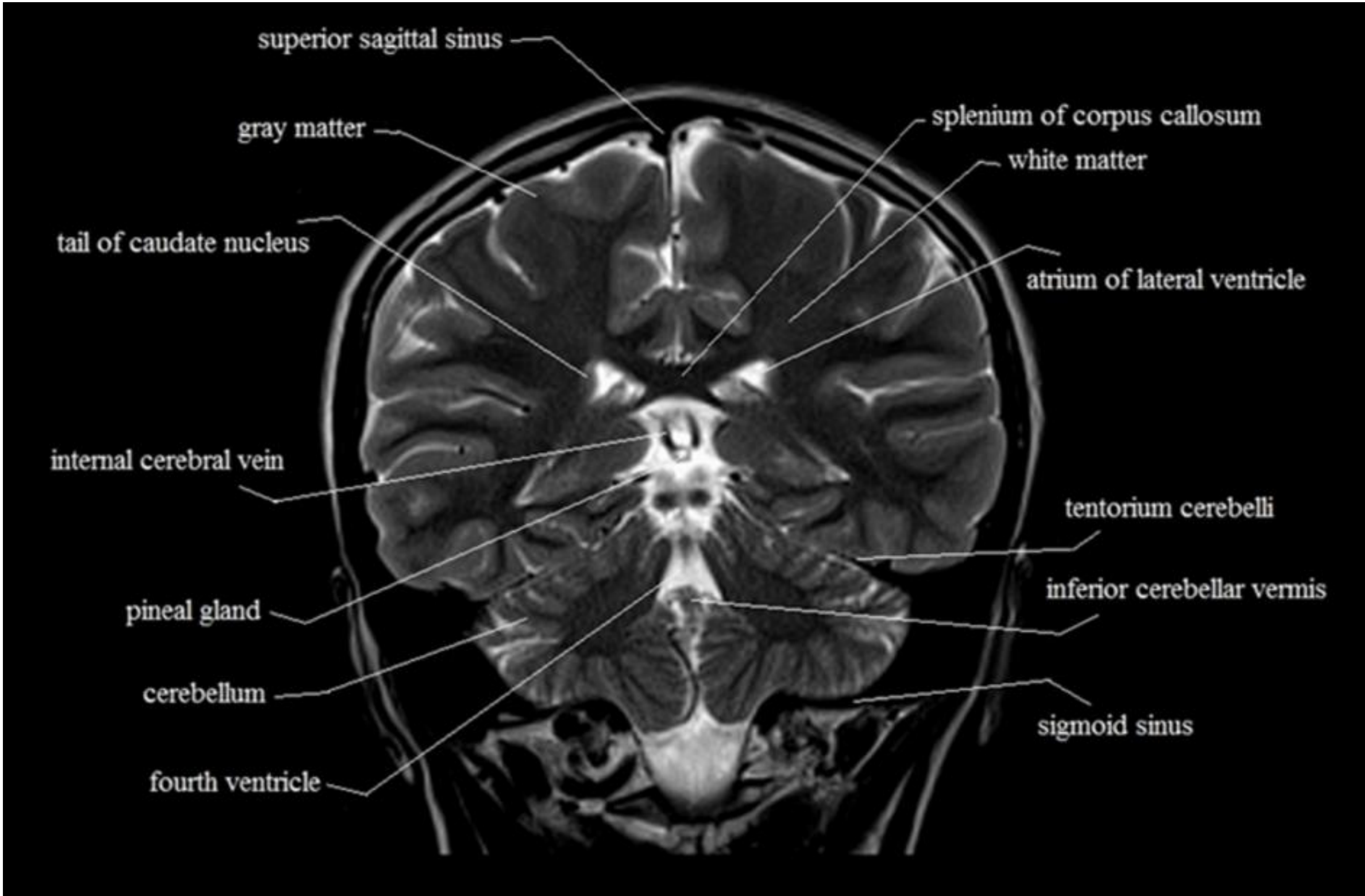
gray matter
body of caudate nucleus
thalamus
internal cerebral vein
hippocampus
pineal gland
cerebellum
fourth ventricle
splenium of corpus callosum
white matter
body of lateral ventricle
cerebral aqueduct
tentorium cerebelli
vertebral artery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gray matter
tail of caudate nucleus
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pineal gland
cerebellum
fourth ventricle
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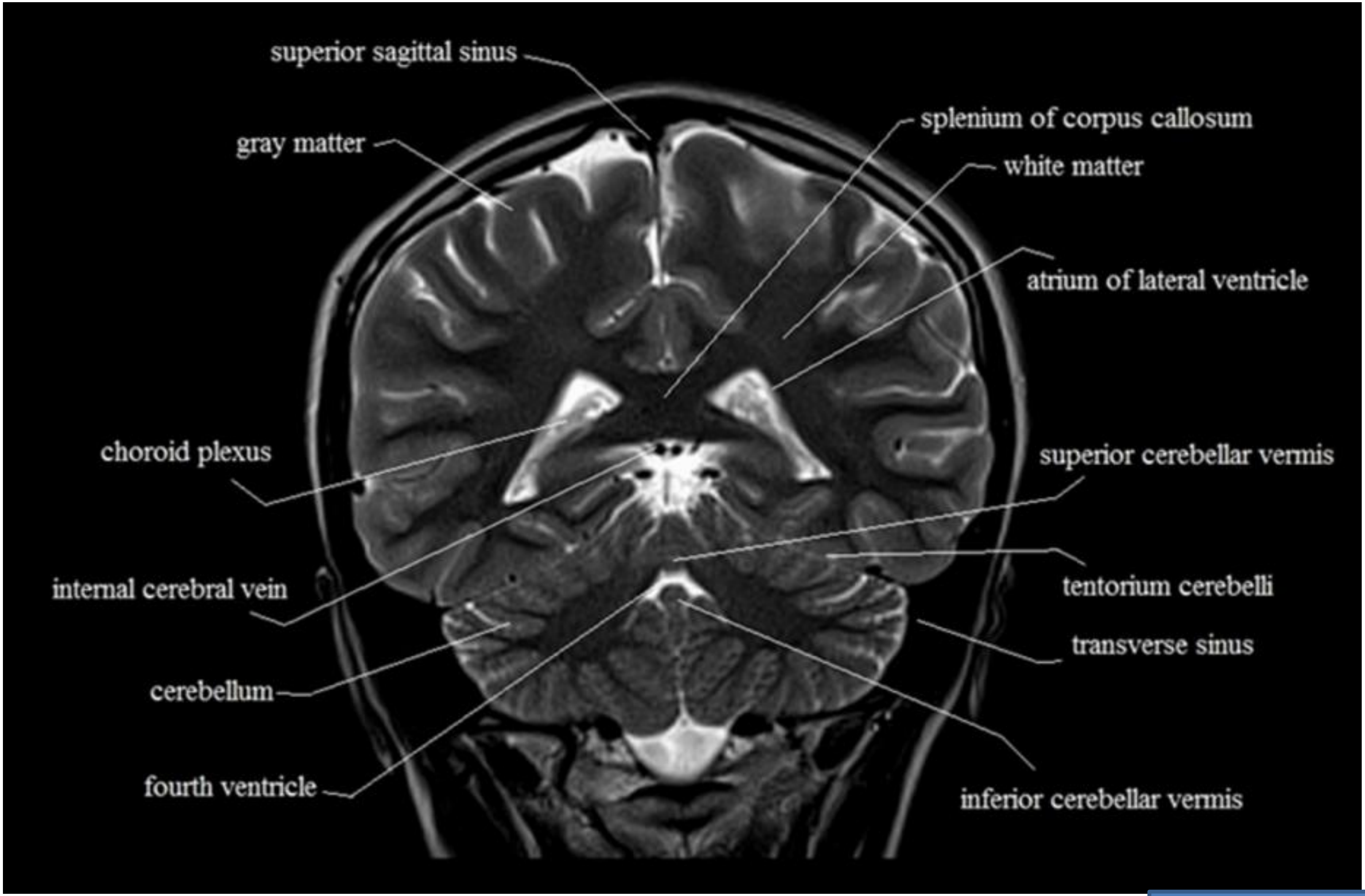
white matter
atrium of lateral ventricle
tentorium cerebelli
inferior cerebellar vermis
sigmoid sinus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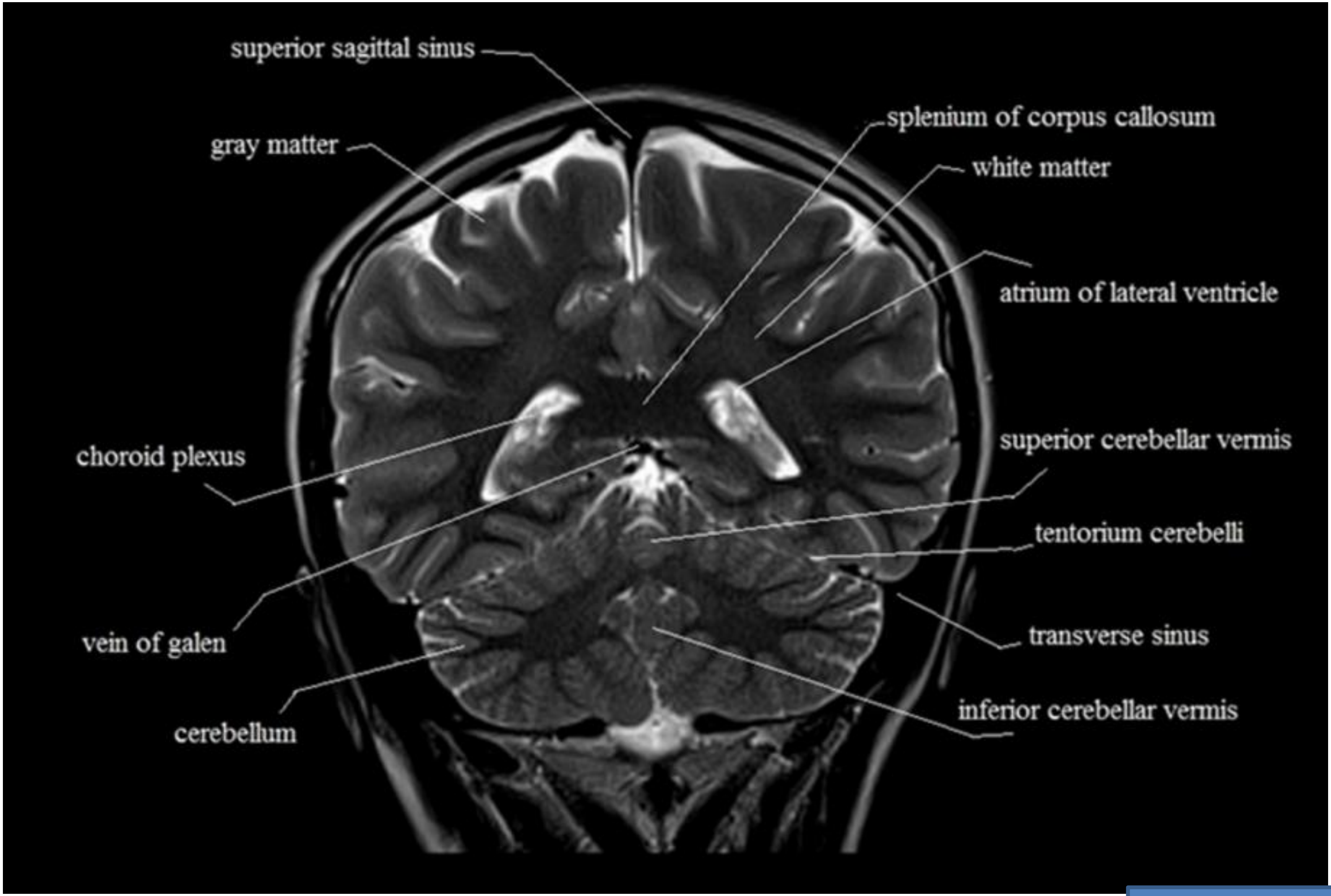
gray matter
splenium of corpus callosum
white matter
atrium of lateral ventricle
choroid plexus
superior cerebellar vermis
internal cerebral vein
tentorium cerebelli
transverse sinus
cerebellum
fourth ventricle
inferior cerebellar vermis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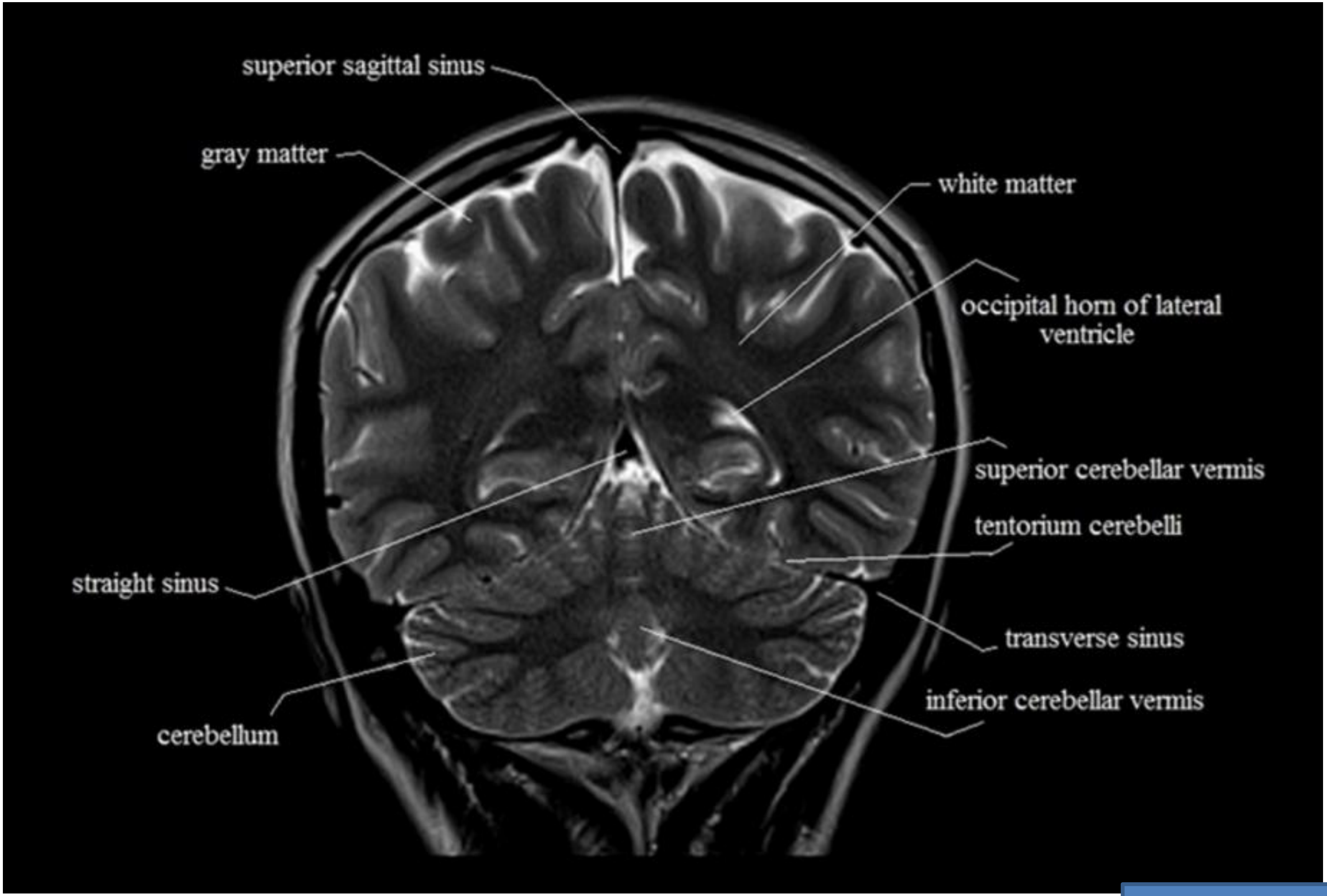
gray matter
splenium of corpus callosum
white matter
atrium of lateral ventricle
choroid plexus
superior cerebellar vermis
tentorium cerebelli
vein of galen
transverse sinus
inferior cerebellar vermis
cerebellum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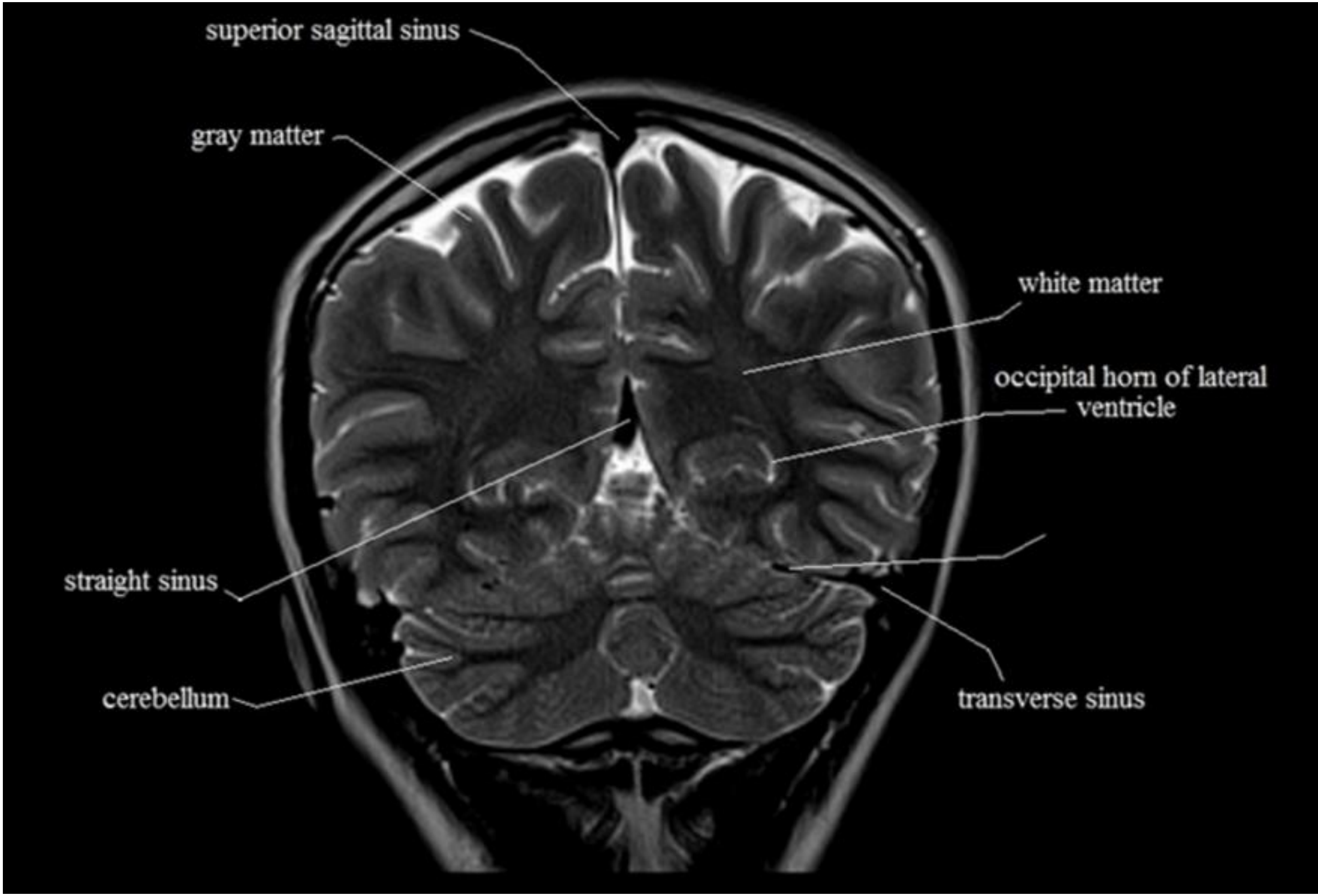
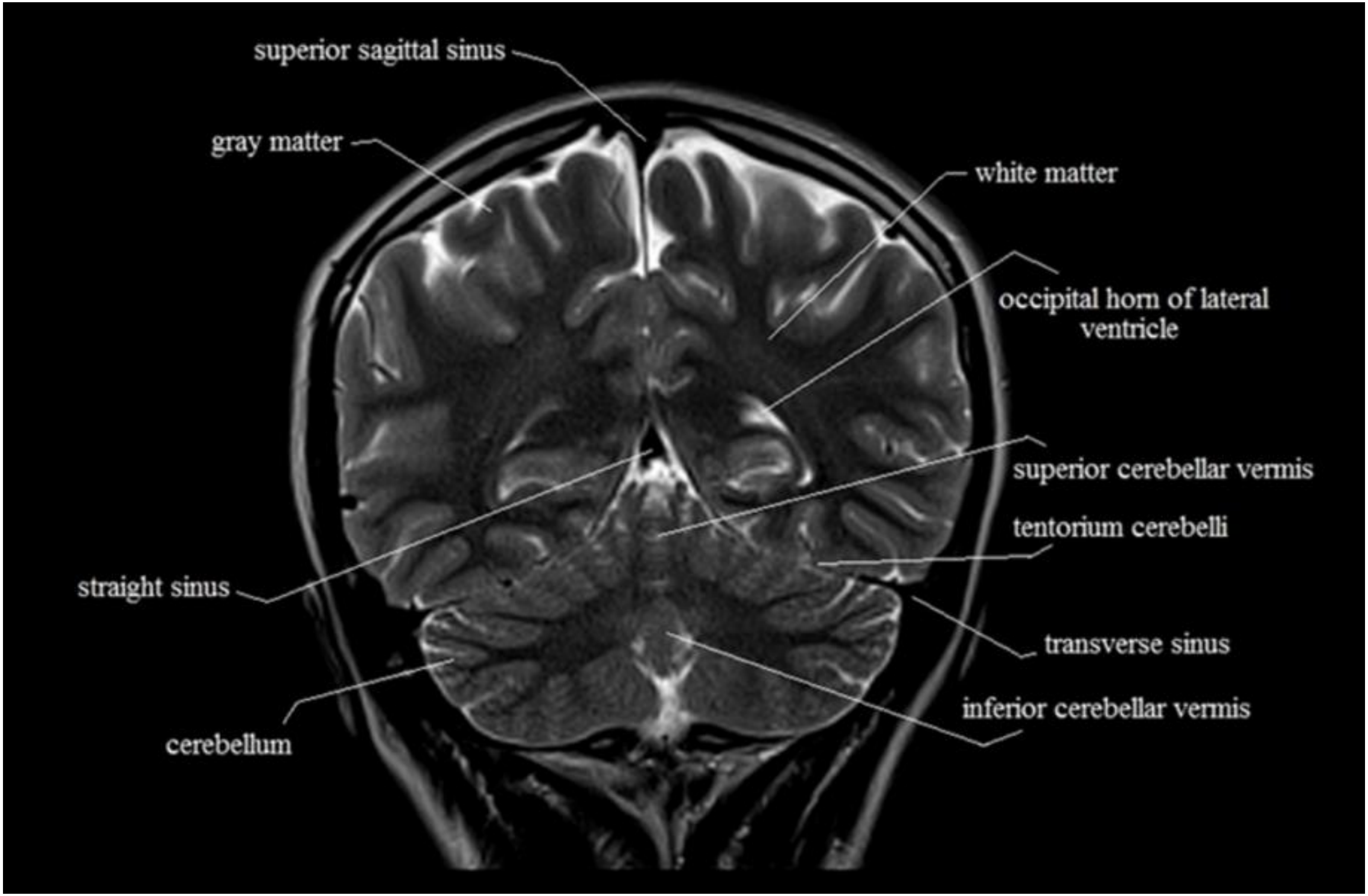
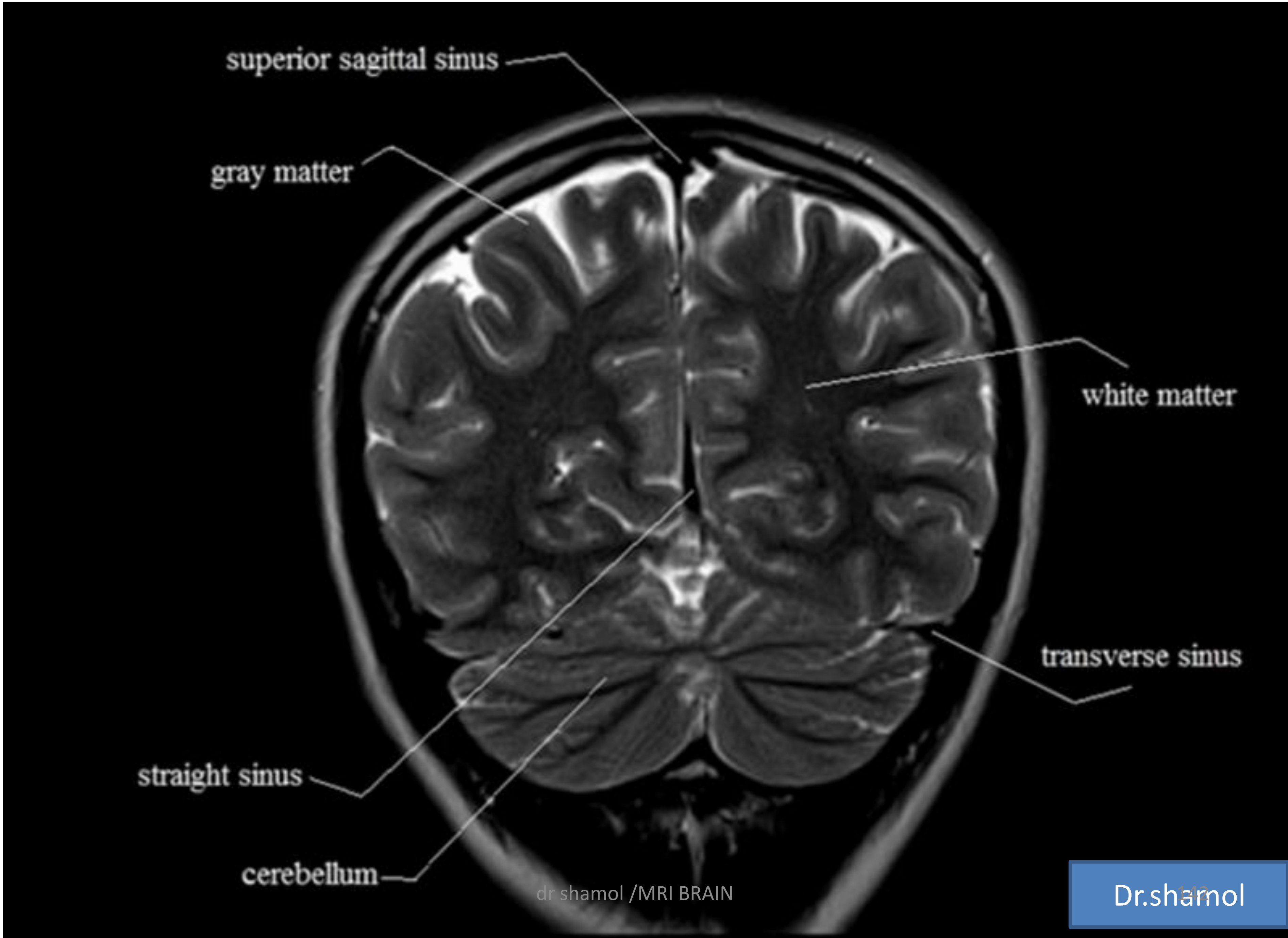
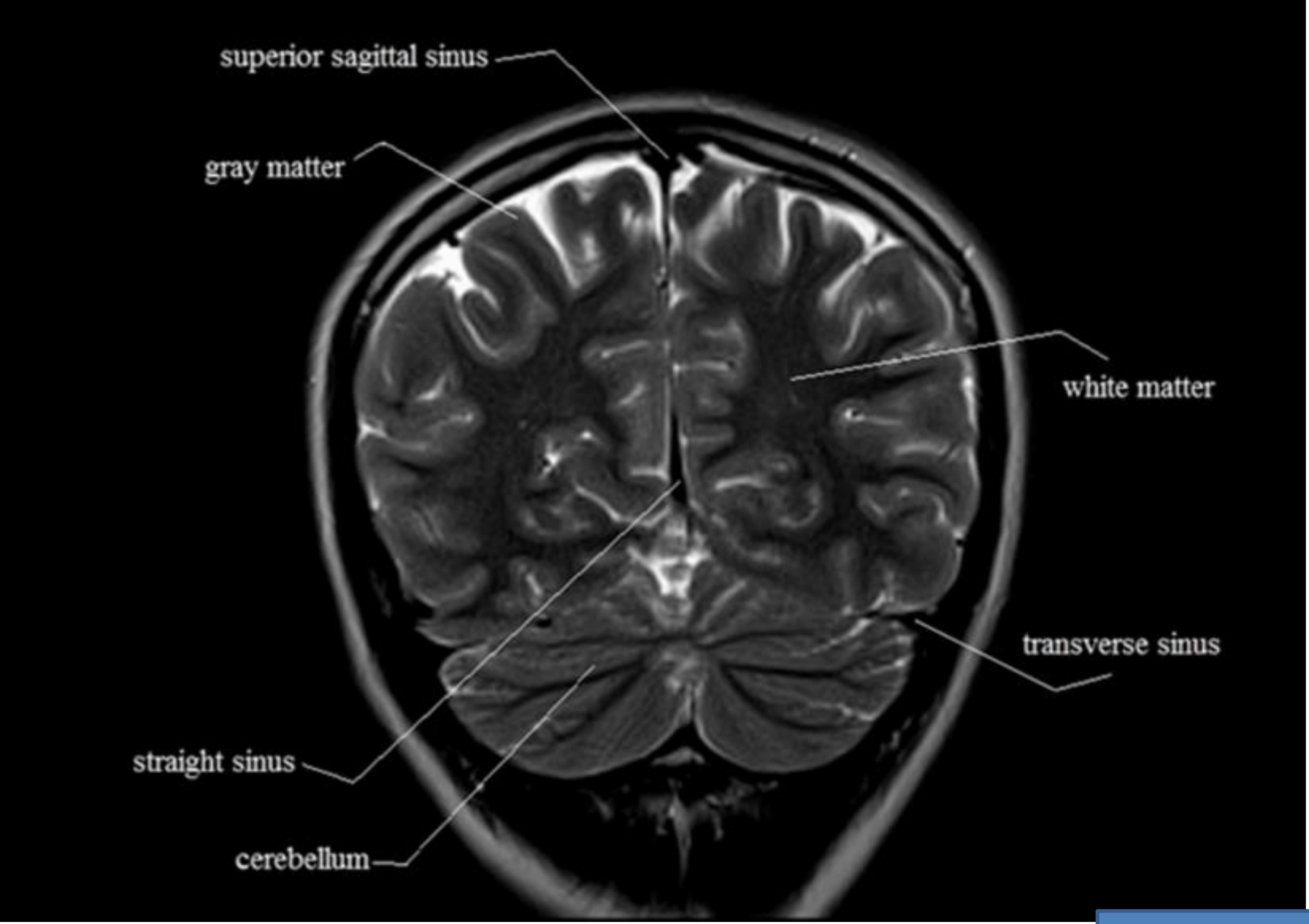
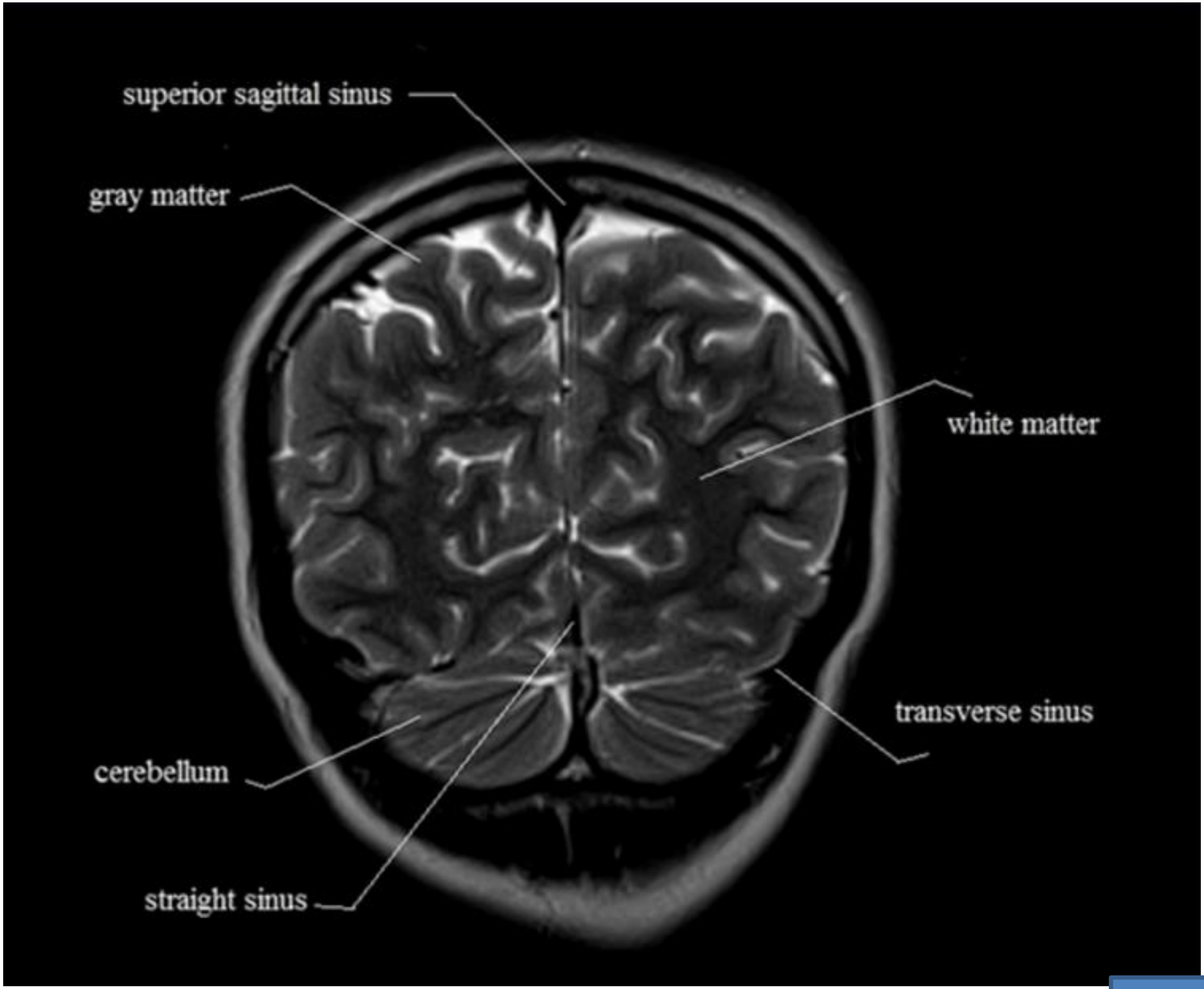
gray matter
white matter
occipital horn of lateral ventricle
superior cerebellar vermis
tentorium cerebelli
straight sinus
transverse sinus
inferior cerebellar vermis
cerebellum

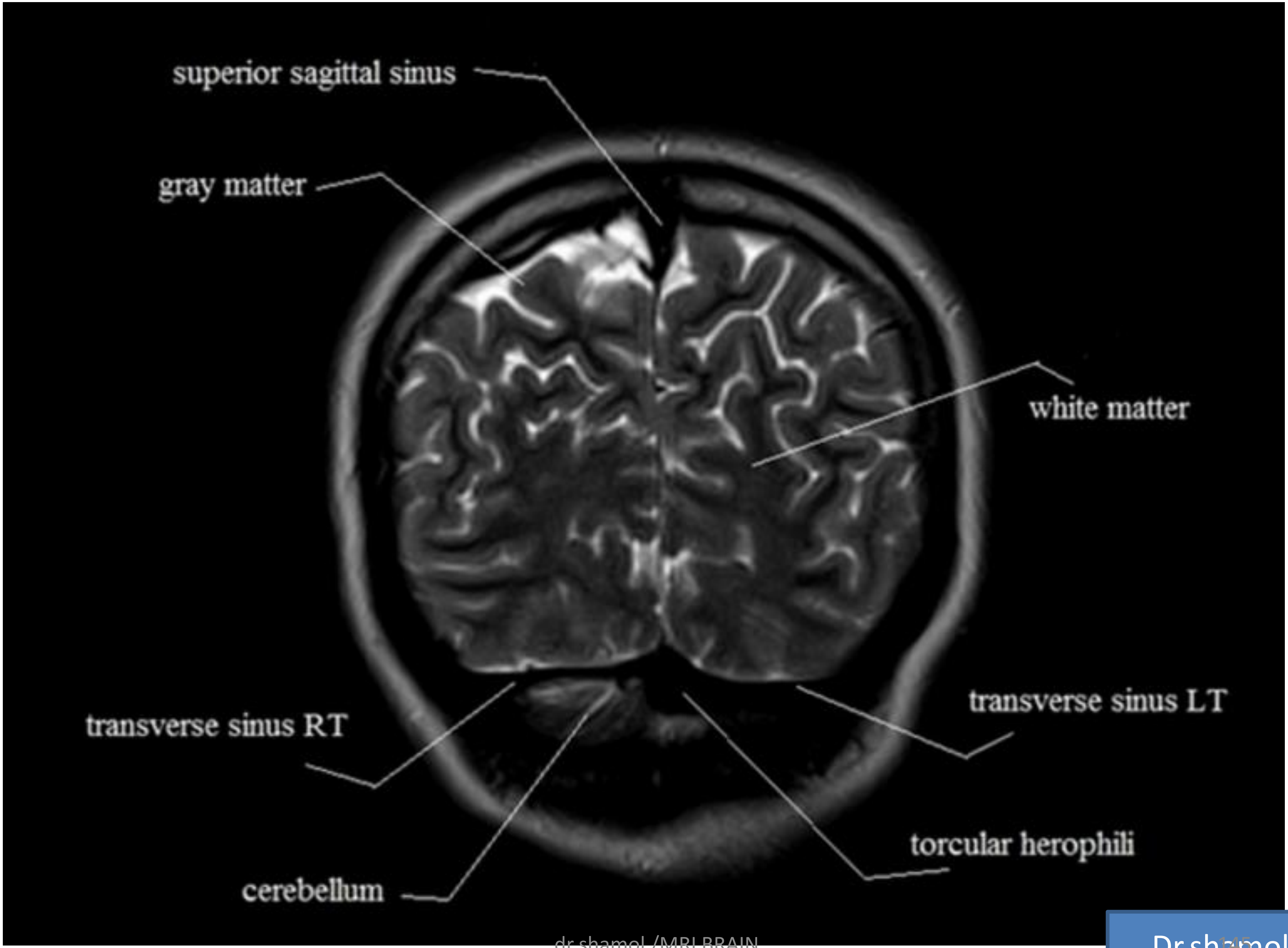

superior sagittal sinus
gray matter
white matter
occipital horn of lateral ventricle
straight sinus
cerebellum
transverse sinus


superior sagittal sinus
gray matter
white matter
occipital horn of lateral ventricle
superior cerebellar vermis
tentorium cerebelli
straight sinus
transverse sinus
inferior cerebellar vermis
cerebellum


superior sagittal sinus
gray matter
white matter
transverse sinus
straight sinus
cerebellum


superior sagittal sinus
gray matter
white matter
transverse sinus
straight sinus
cerebellum


superior sagittal sinus
gray matter
white matter
transverse sinus
cerebellum
straight sinus


superior sagittal sinus
gray matter
white matter
transverse sinus LT
transverse sinus RT
torcular herophili
cerebellum

NOW IDENTIFY THE STRUCTURE'S BY YOUR SELF

ROW -1

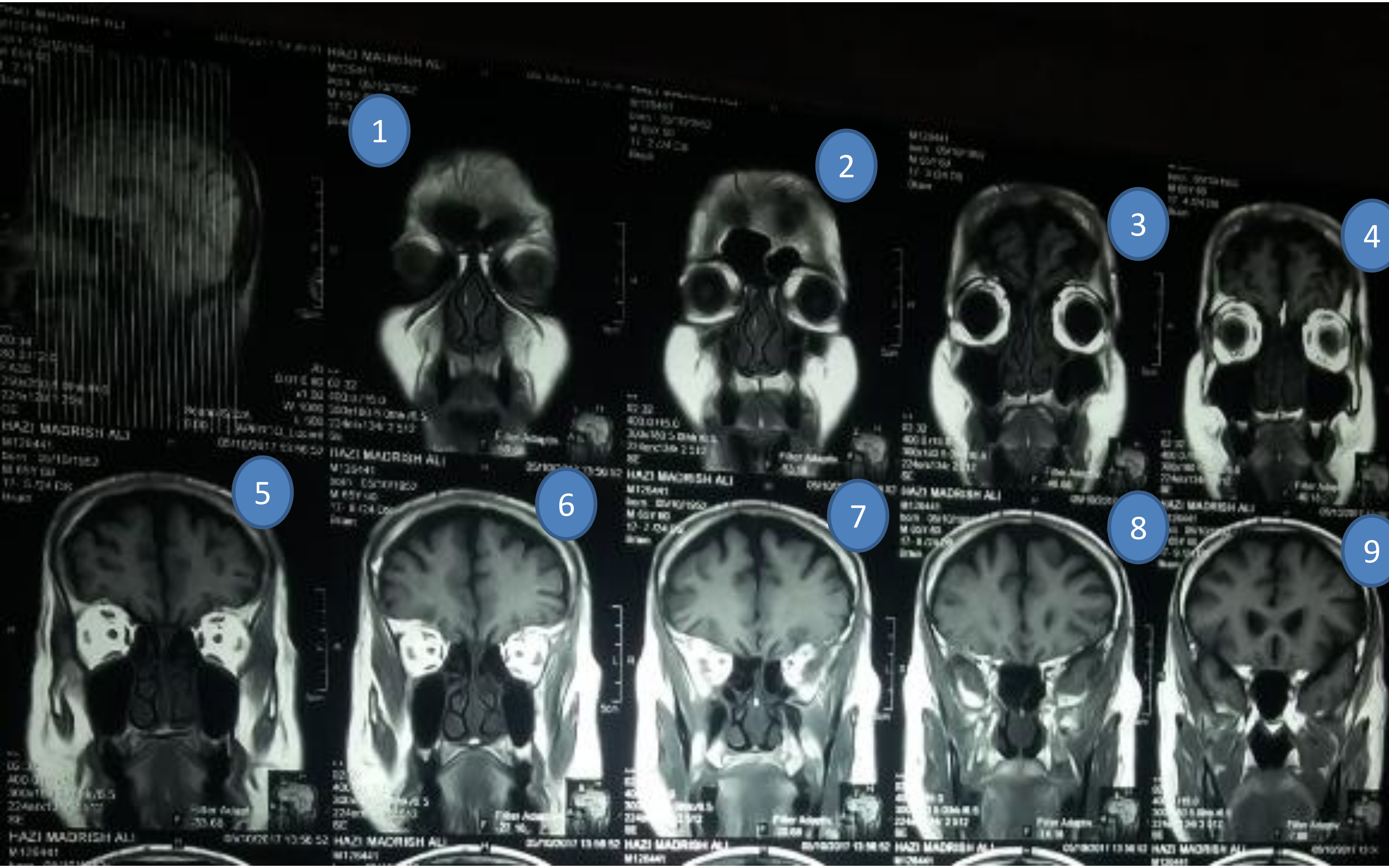
ROW -2

ROW -3

ROW -4

ROW -5

Dr.shamol


1
2
3
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7
8
9


10
11
12
13
!4
15
16
17
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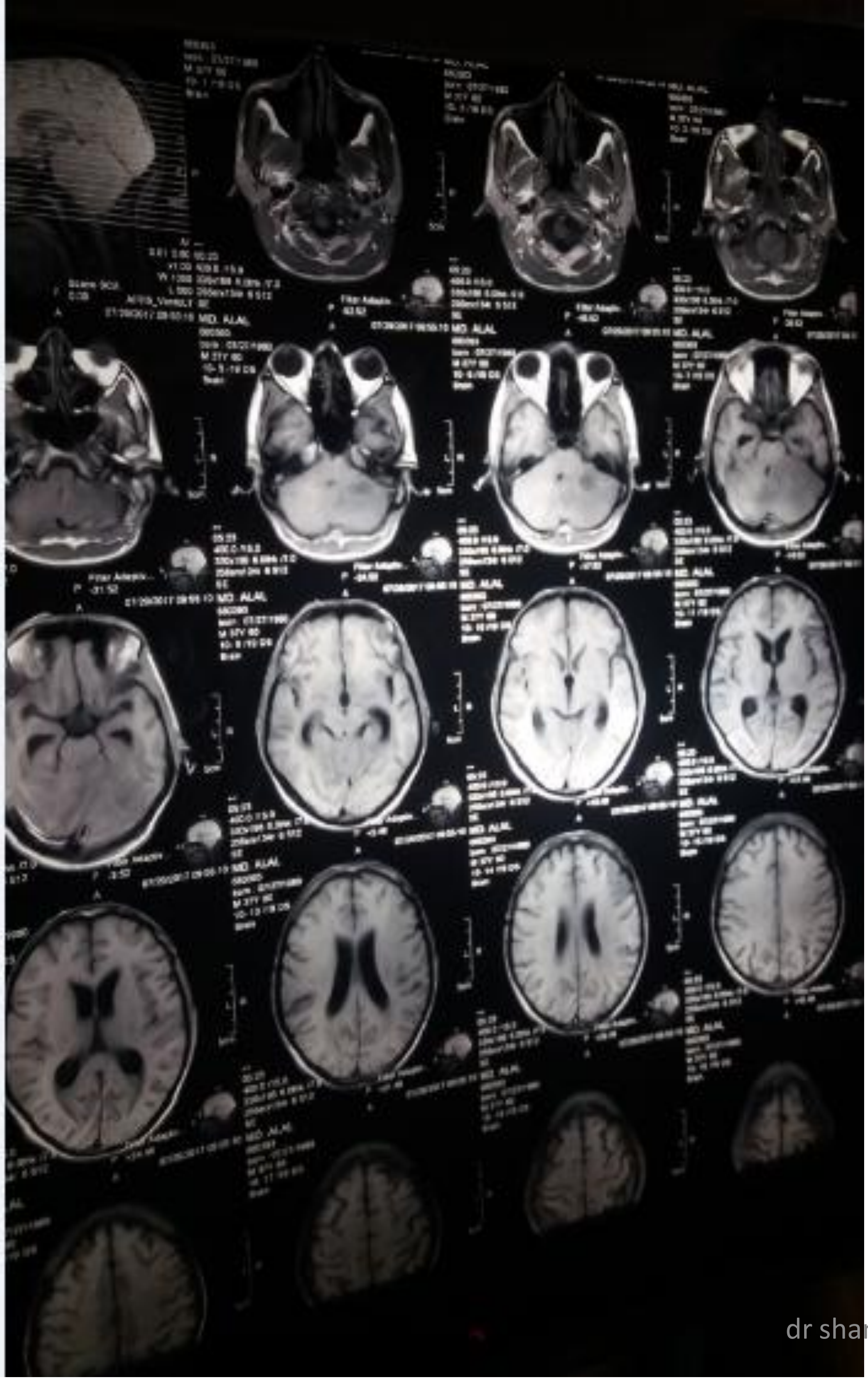
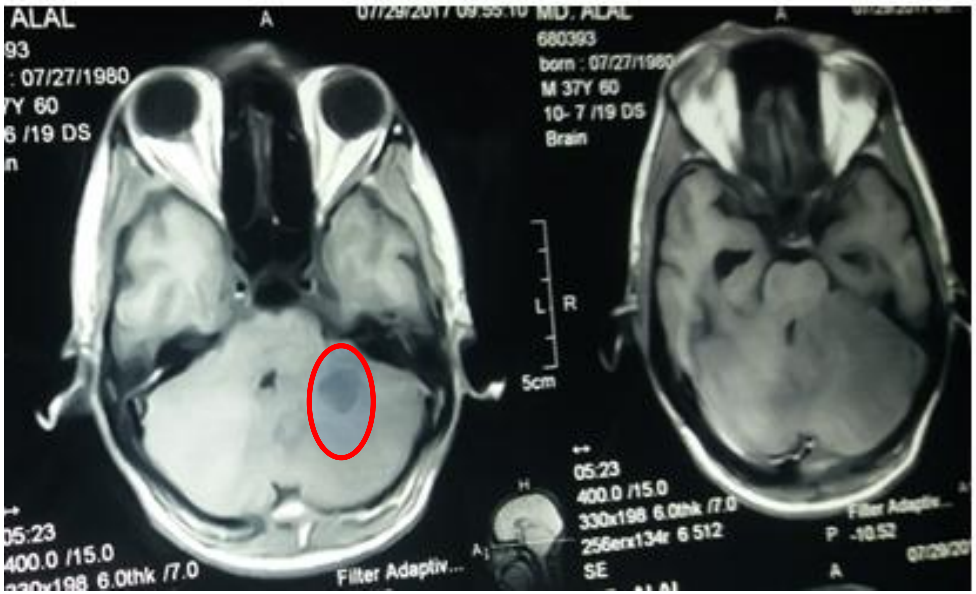
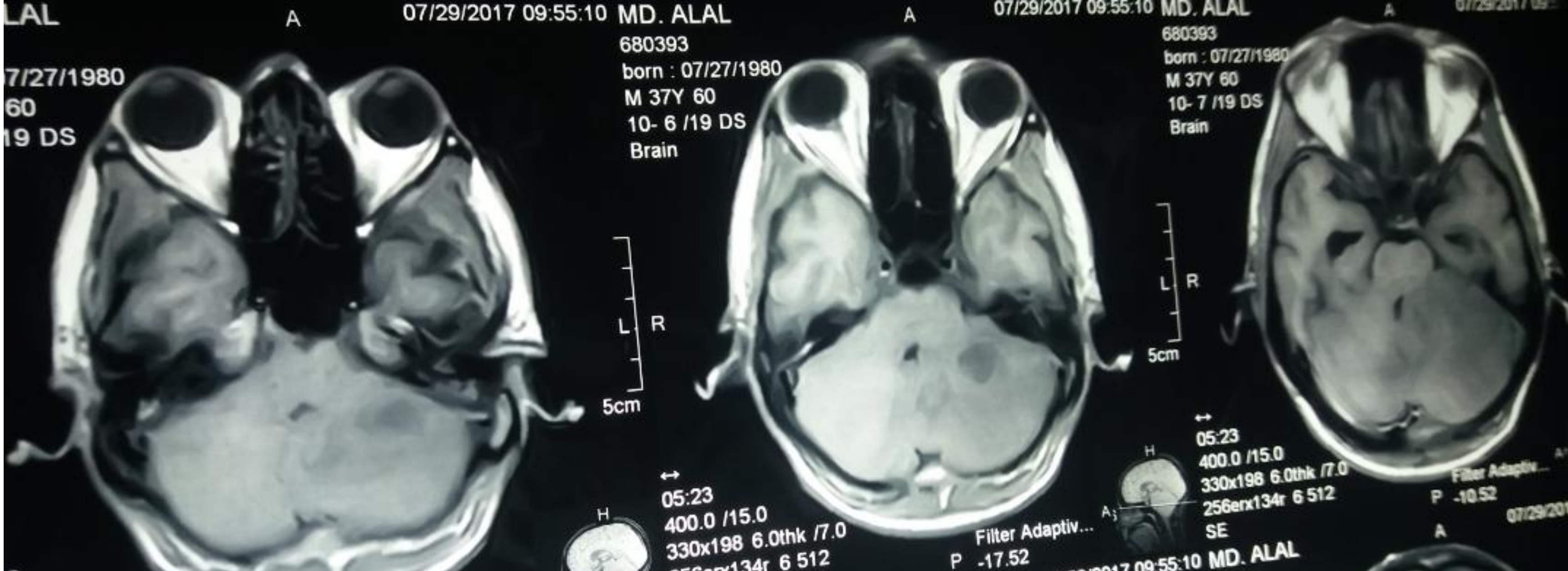
21
22
23
24

Lesion in MRI



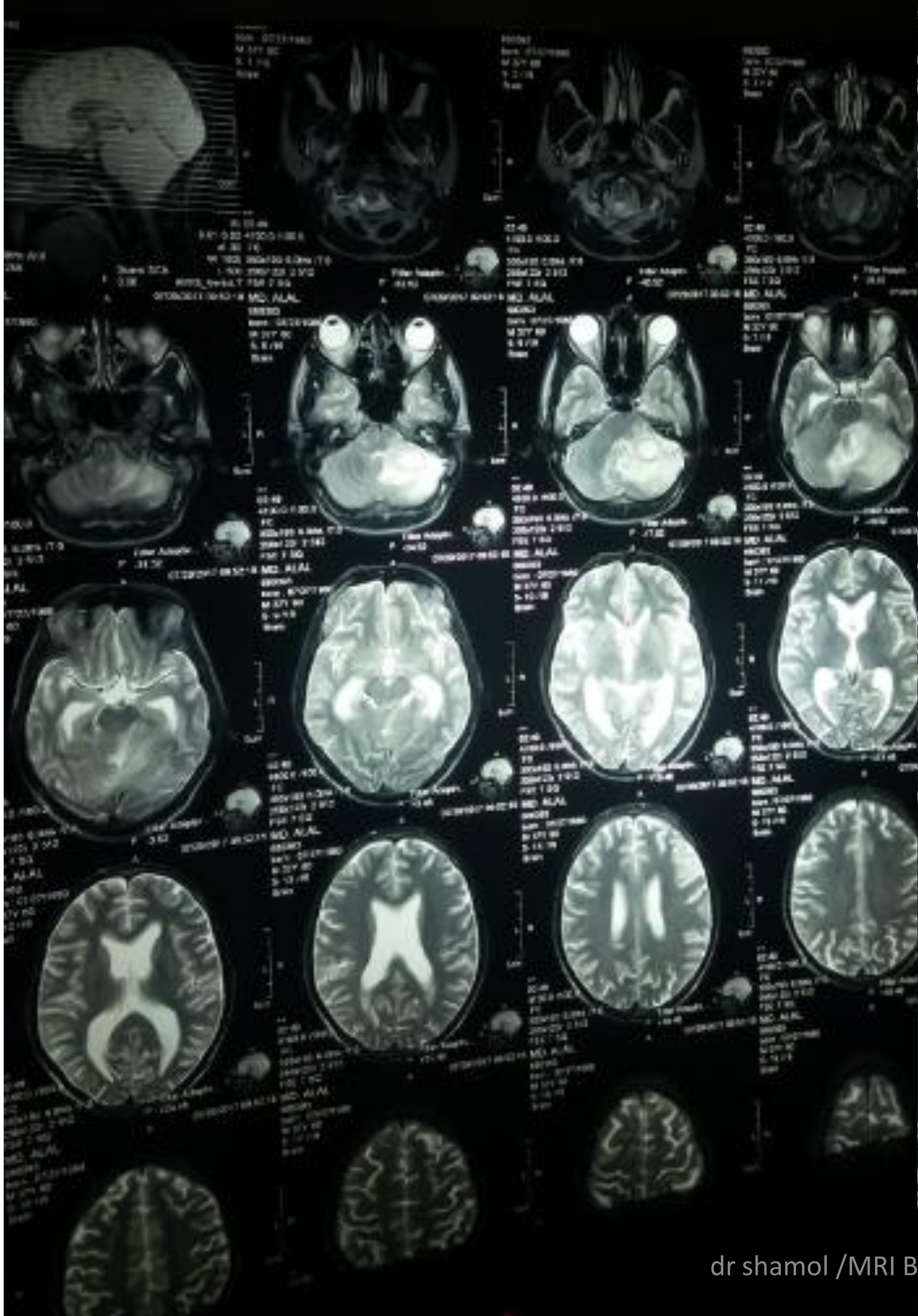
Hypointens
(black) lesion
In brain
In T1



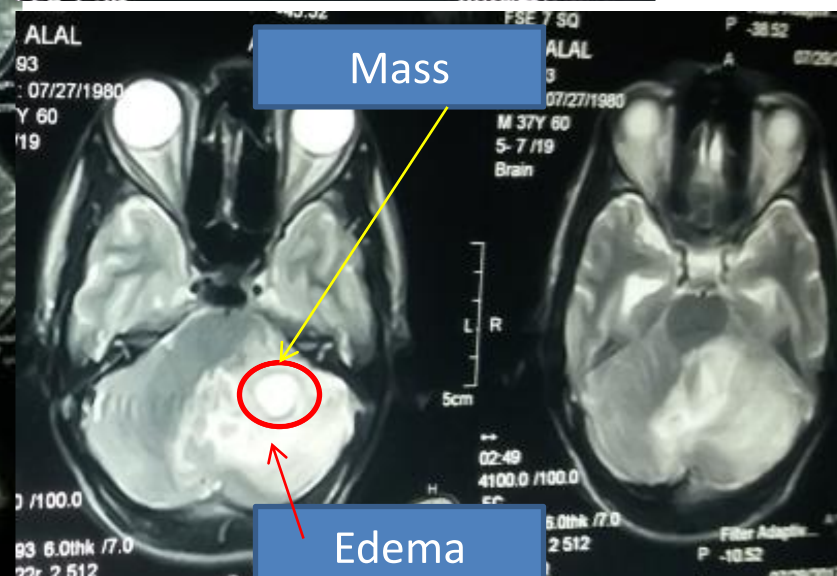

07/29/2017 09:55:10 MD. ALAL
680393
born : 07/27/1980
M 37Y 60
10- 6 /19 DS
Brain
A
L R
5cm
05:23
400.0 /15.0
330x198 6.0thk /7.0
Filter Adaptiv...
P -17.52
07/29/2017 09:55:10 MD. ALAL
680393
born : 07/27/1980
M 37Y 60
10- 7 /19 DS
Brain
05:23
400.0 /15.0
330x198 6.0thk /7.0
256erx134r 6 512
SE
P -10.52


:23
0.0 /15.0
6.0thk /7.0
Filter Adaptiv...

Dr.shamol



Hyper-intens
(white)
In T2



Dr.shamol

MD. ALAL
680393
born : 07/27/1980
M 37Y 60
5- 5 /19
Brain
P -52.52
07/29/2017 09:52:18
R
L
5cm
02:49
4100.0 /100.0
FC
256x122r 2 512
FSE 7 SQ
MD. ALAL
680393
born : 07/27/1980
M 37Y 60
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Brain
Filter Adaptiv...
P -45.52
07/29/2017 09:52:18
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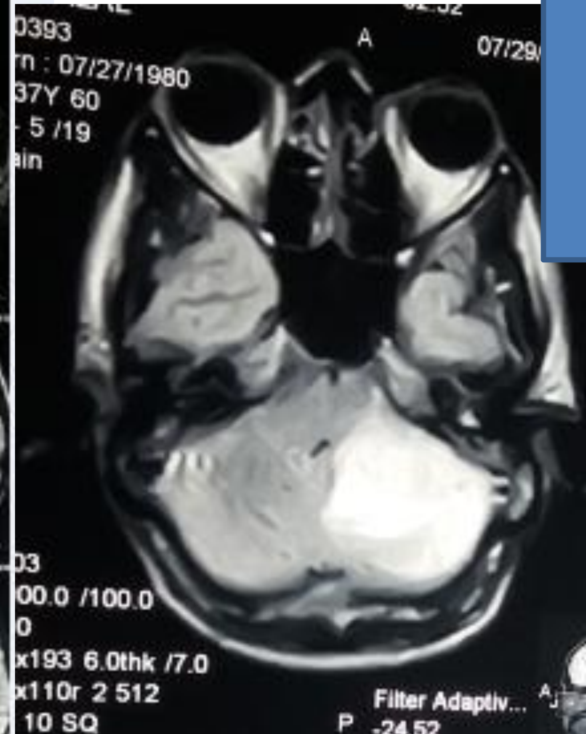
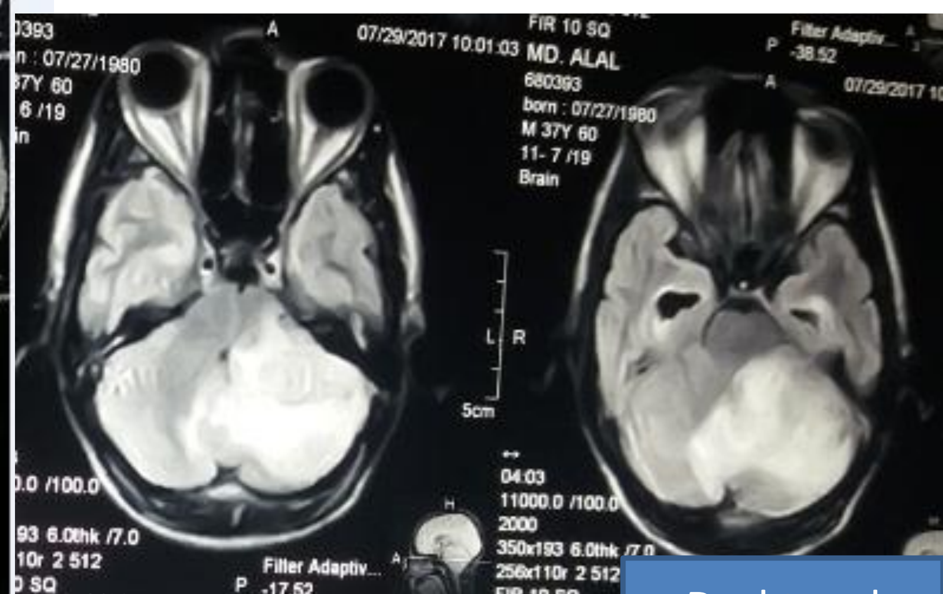
256x122r 2 512
Filter Adaptiv...
256x122r 2 512
FSE 7 SQ
MD. ALAL
680393
born : 07/27/1980
M 37Y 60
5- 7 /19
Brain
R
L
5cm
02:49
4100.0 /100.0
FC
350x193 6.0thk /7.0
256x122r 2 512
FSE 7 SQ

L
5cm
2:49
100.0 /100.0
FC
50x193 6.0thk /7.0
Filter Adaptiv...
H

Dr.shamol



Hyper-intens
(white)
In FLAIR



Dr.shamol


MD. ALAL
680393
born : 07/27/1980
M 37Y 60
11- 5 /19
Brain
07/29/2017 10:01:03
04:03
11000.0 /100.0
2000
350x193 6.0thk /7.0
256x110r 2 512
FIR 10 SQ
MD. ALAL
680393
born : 07/27/1980
M 37Y 60
11- 6 /19
Brain
Filter Adaptiv...
P -45.52
5cm
07/29/2017 10:01:03
MD. ALAL
680393
born : 07/27/1980
M 37Y 60
11- 7 /19
Brain
350x193 6.0thk /7.0
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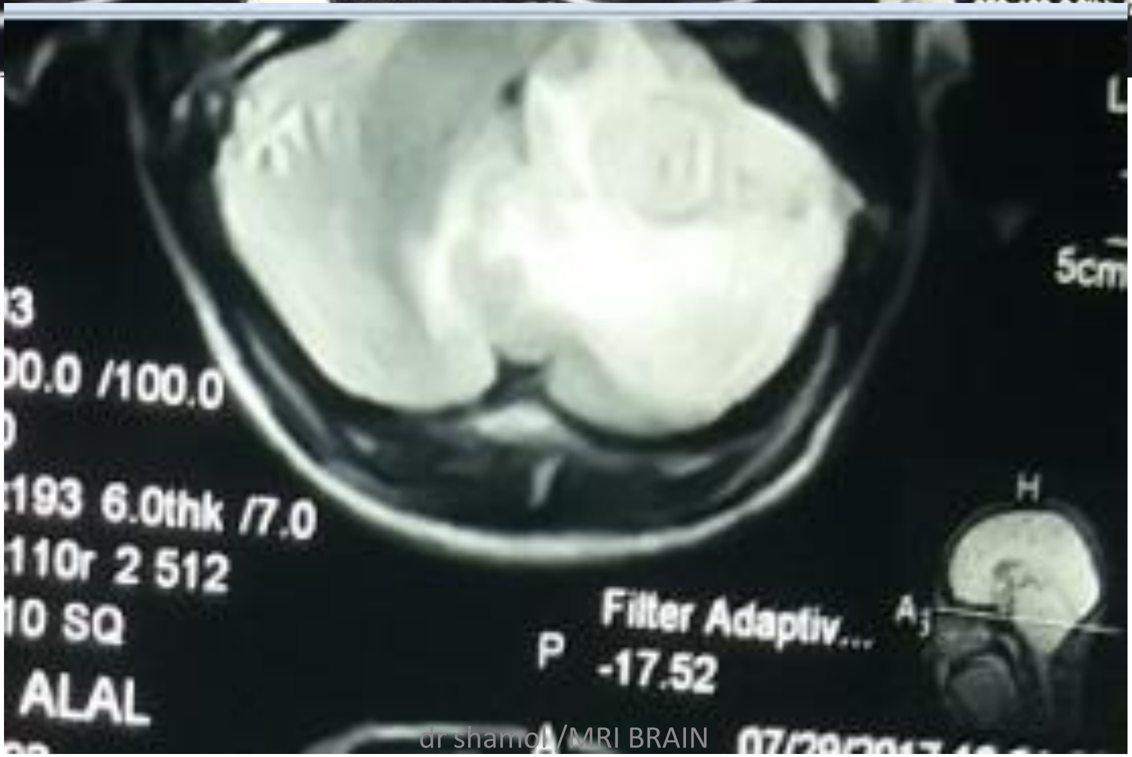
FIR 10 SQ
Filter Adaptiv...
P -38.52
Filter Adaptiv...
P -10.52

00.0 /100.0
193 6.0thk /7.0
110r 2 512
10 SQ
ALAL
Filter Adaptiv...
P -17.52
5cm

Dr.shamol


T 1
393
n : 07/27/1980
37Y 60
- 6 /19 DS
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R
L
5cm
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400.0 /15.0
230x198 6.0thk /7.0
Filter Adaptiv...
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flair
D. ALAL
0393
rn : 07/27/1980
37Y 60
- 6 /19
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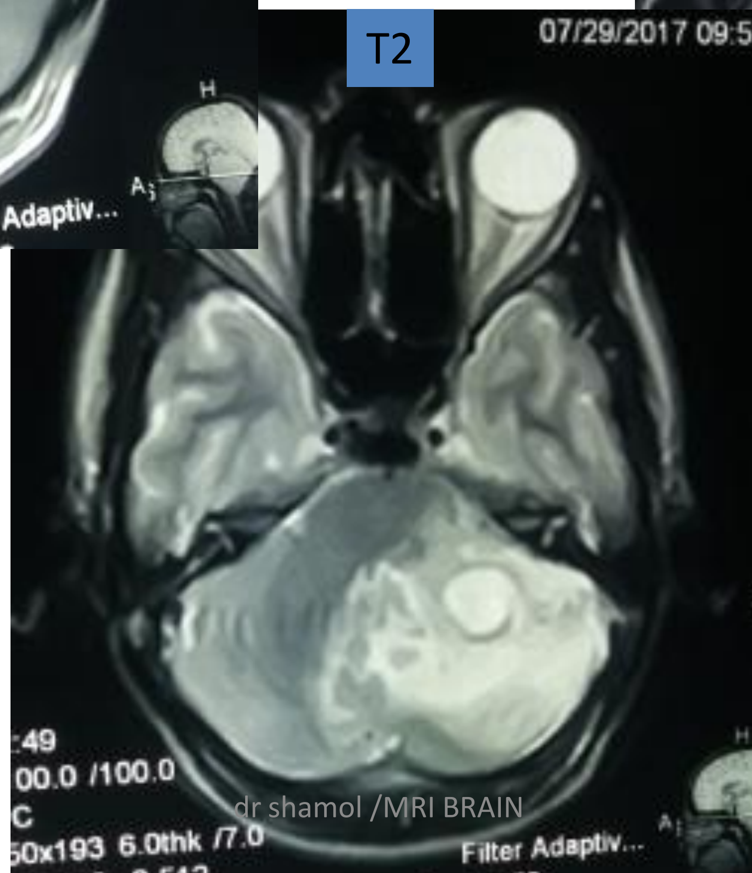
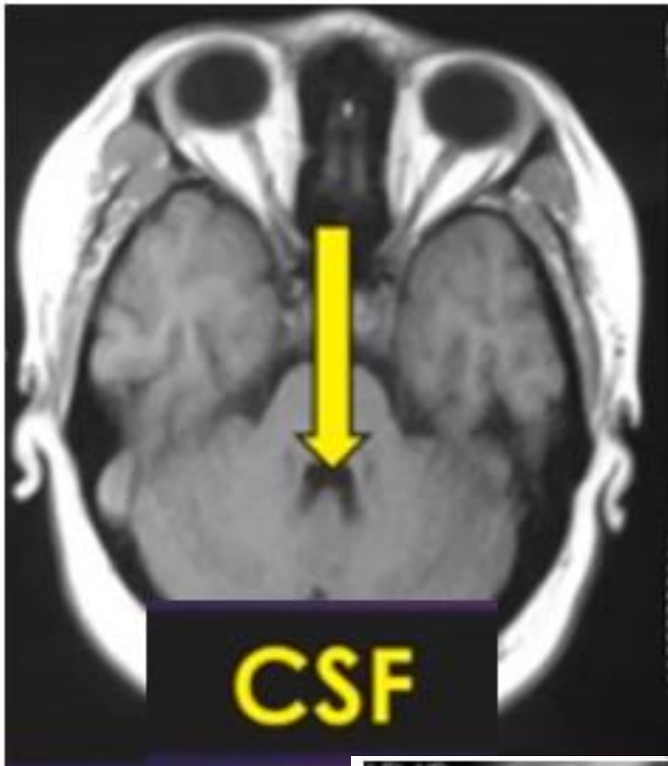
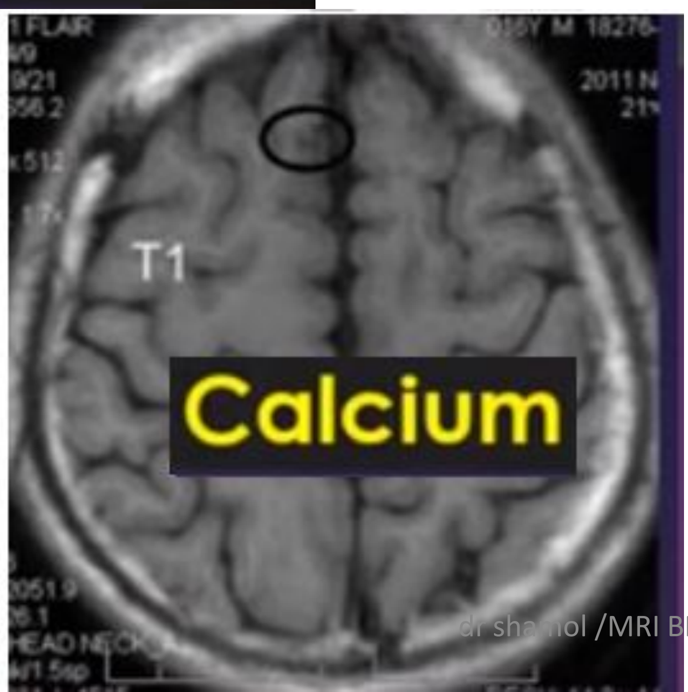

T2
07/29/2017 09:52
:49
00.0 /100.0
C
50x193 6.0thk /7.0
Filter Adaptiv...
H

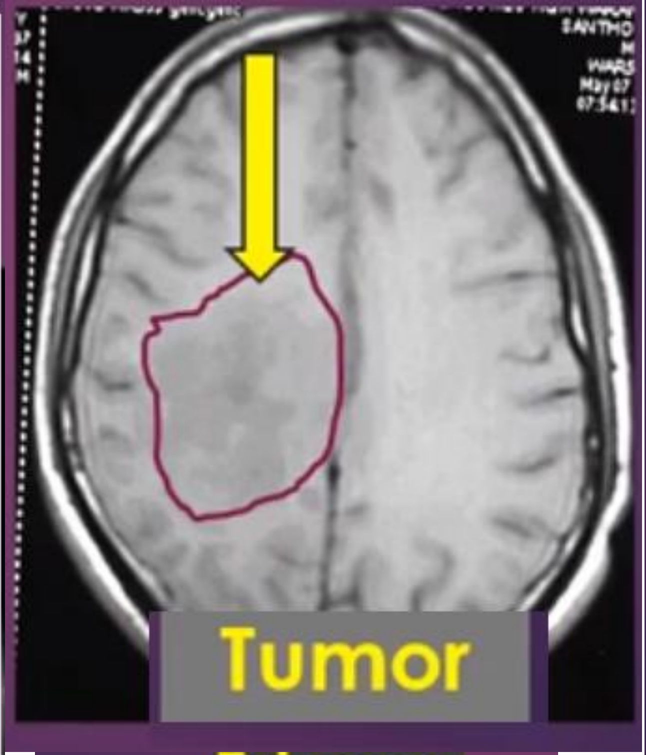
Structures look grey
&
Structures look dark
Structures look white
In T1

Dr.shamol

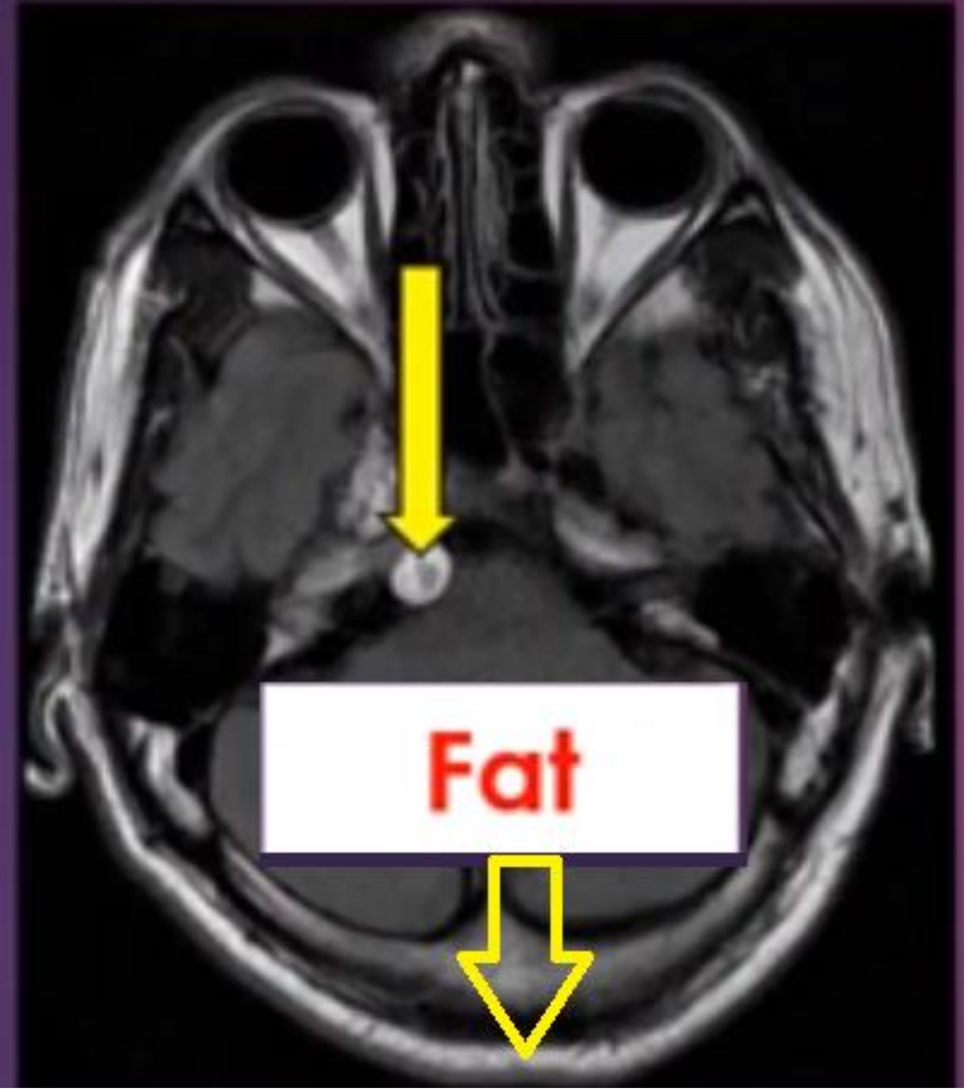


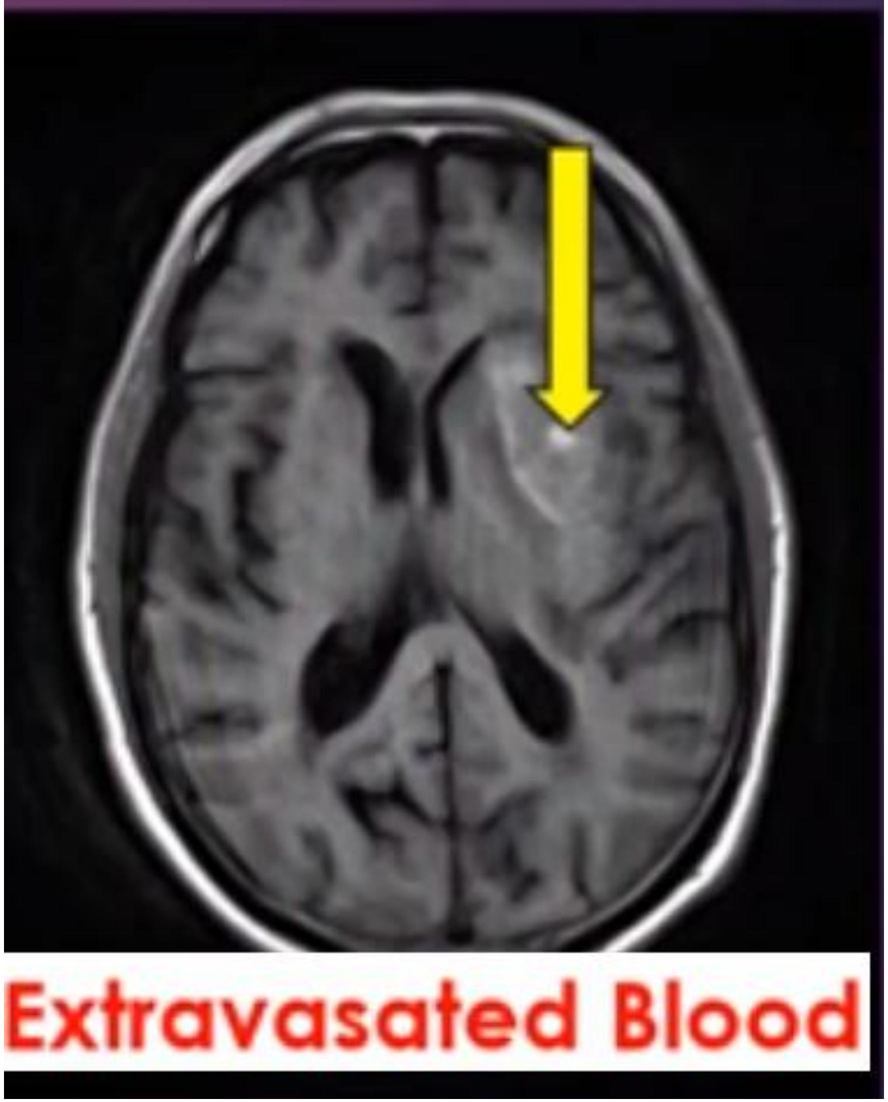
Structure that Look black



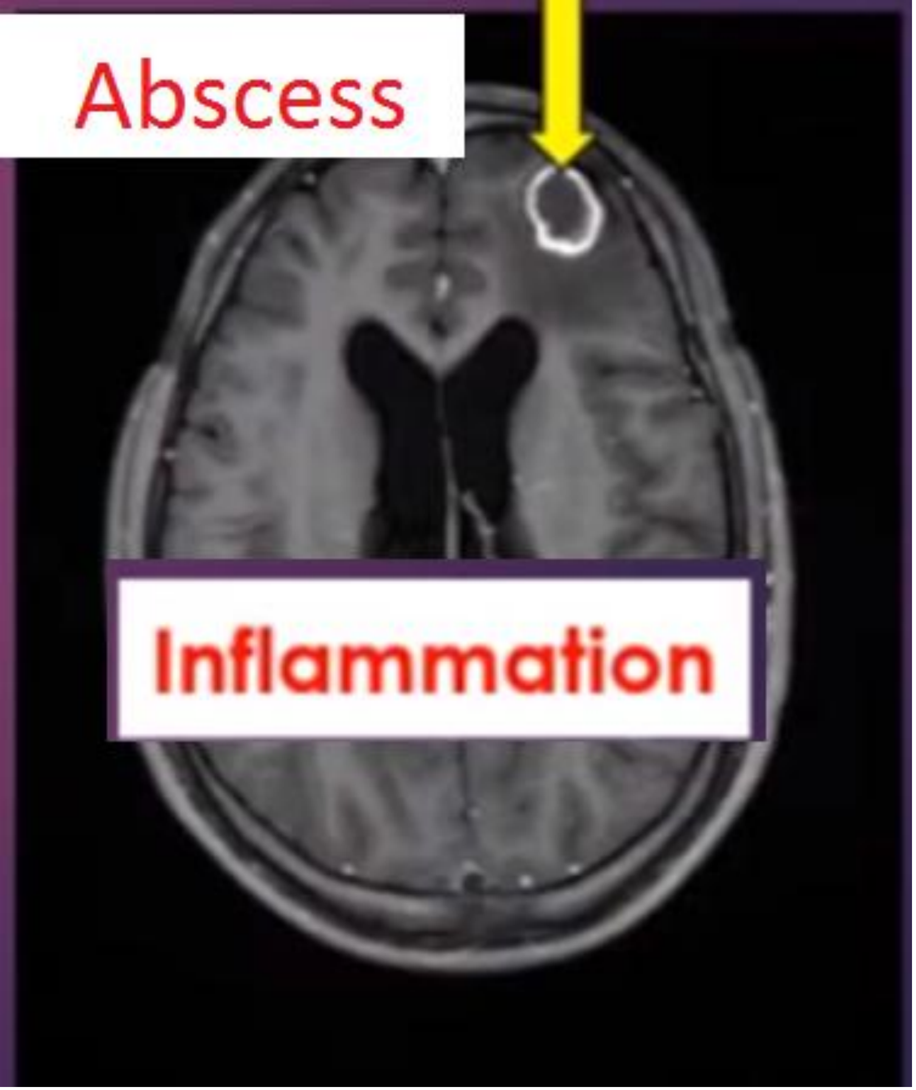


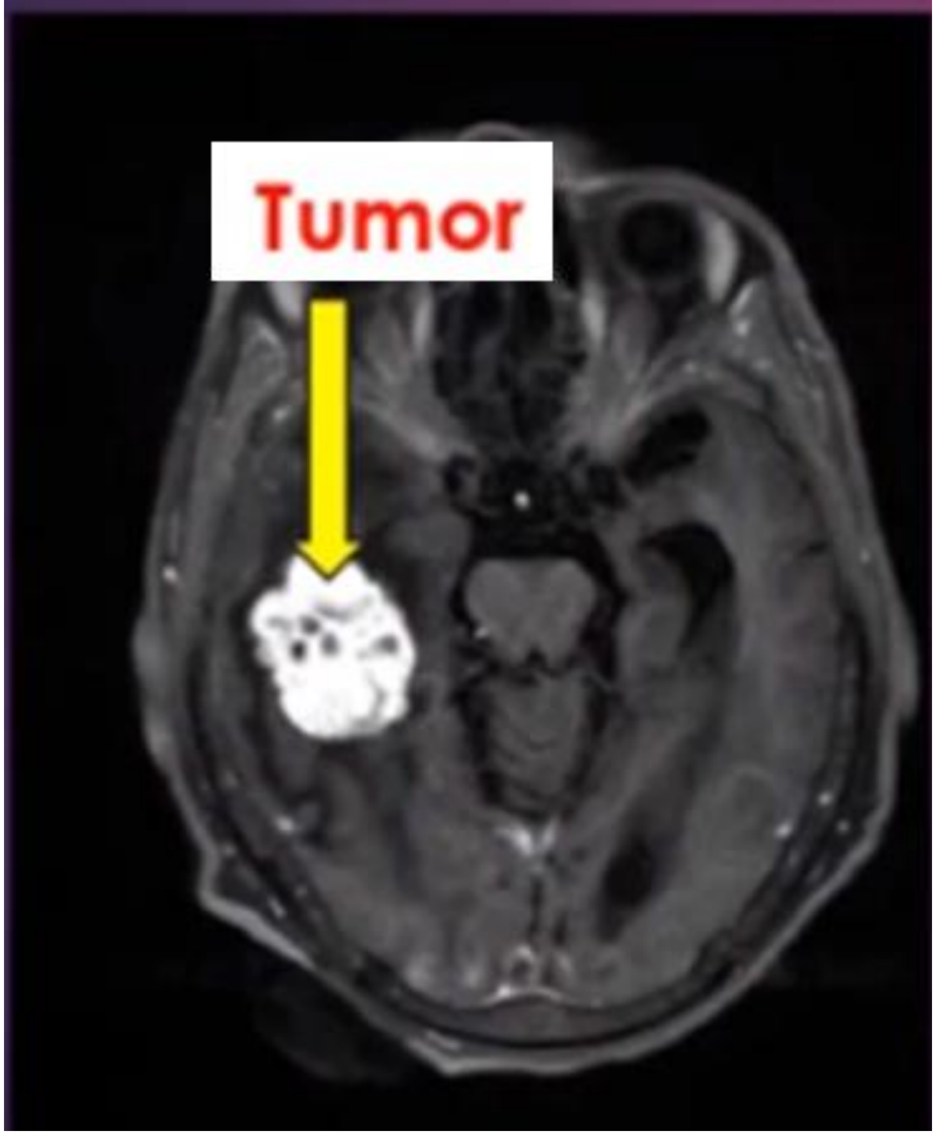
Structures look grey





Structure that look white



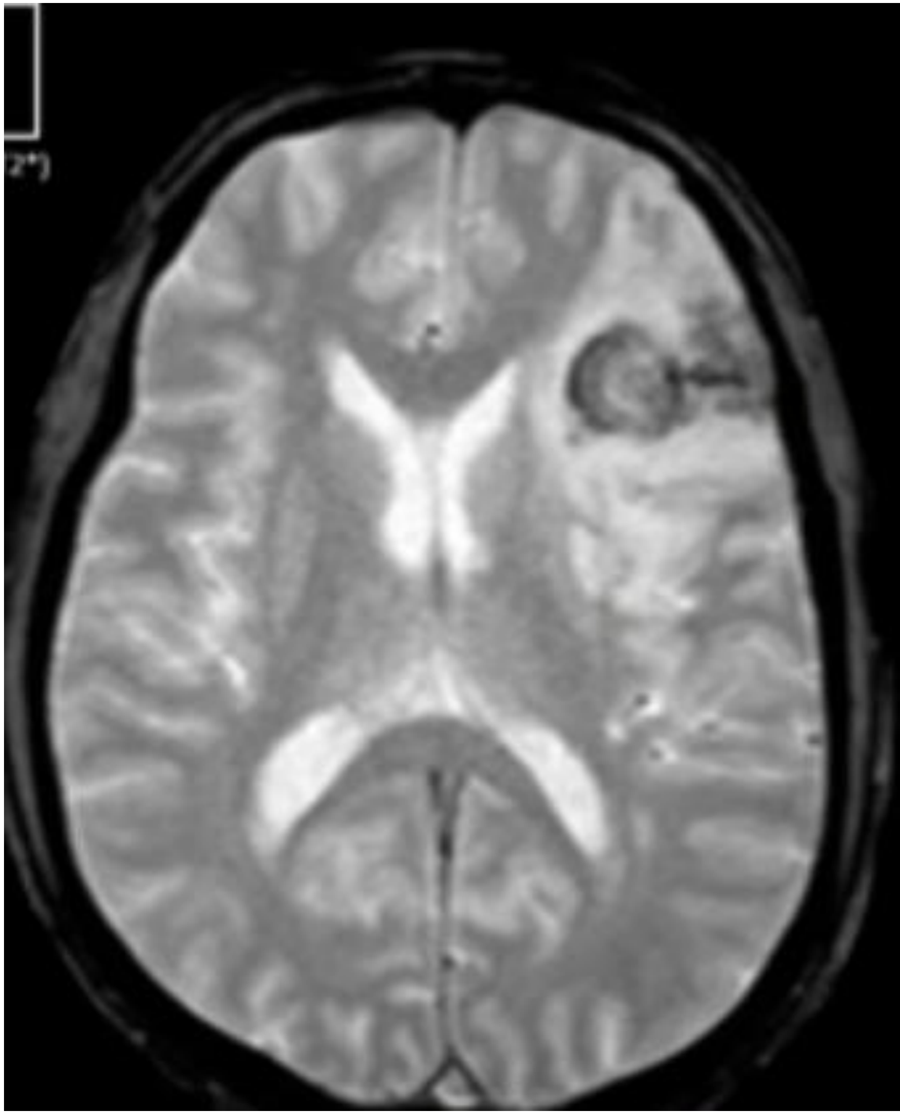


Contrast enhance

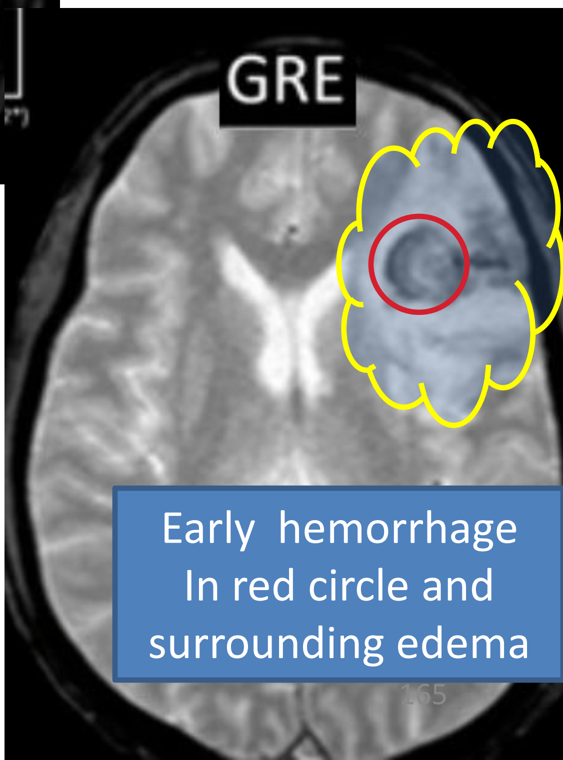
STRUCTURE THAT LOOK DARK IN T2

Like T 2
Paramagnetic substances are dark
Blood
Calcium
Other metal (cupper in wilson)

Useful for
Early hemorrhage
Old hemorrhage (hemosiderin)



Its look like edema



Early hemorrhage
In red circle and
surrounding edema

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Contrast enhancement

Extra-axial tumors such as meningiomas and schwannomas

High grade gliomas -

- astrocytoma
- oligodendrogliomas
- glioblastoma multiforme

Tumour of pituitary, pineal and choroid plexus

non-tumoral lesions

- abscess
- Infections (Tuberculoma)
- Demyelinating diseases (MS)
- infarctions

Low grade gliomas not enhance expect this two

- Gangliogliomas
- Gpilocytic astrocytomas

Dr.shamol

Contrast **Enhancement**

❖The brain has a unique triple layered blood-brain barrier (BBB) with tight endothelial junctions Contrast will not leak into the brain unless this barrier is damaged.

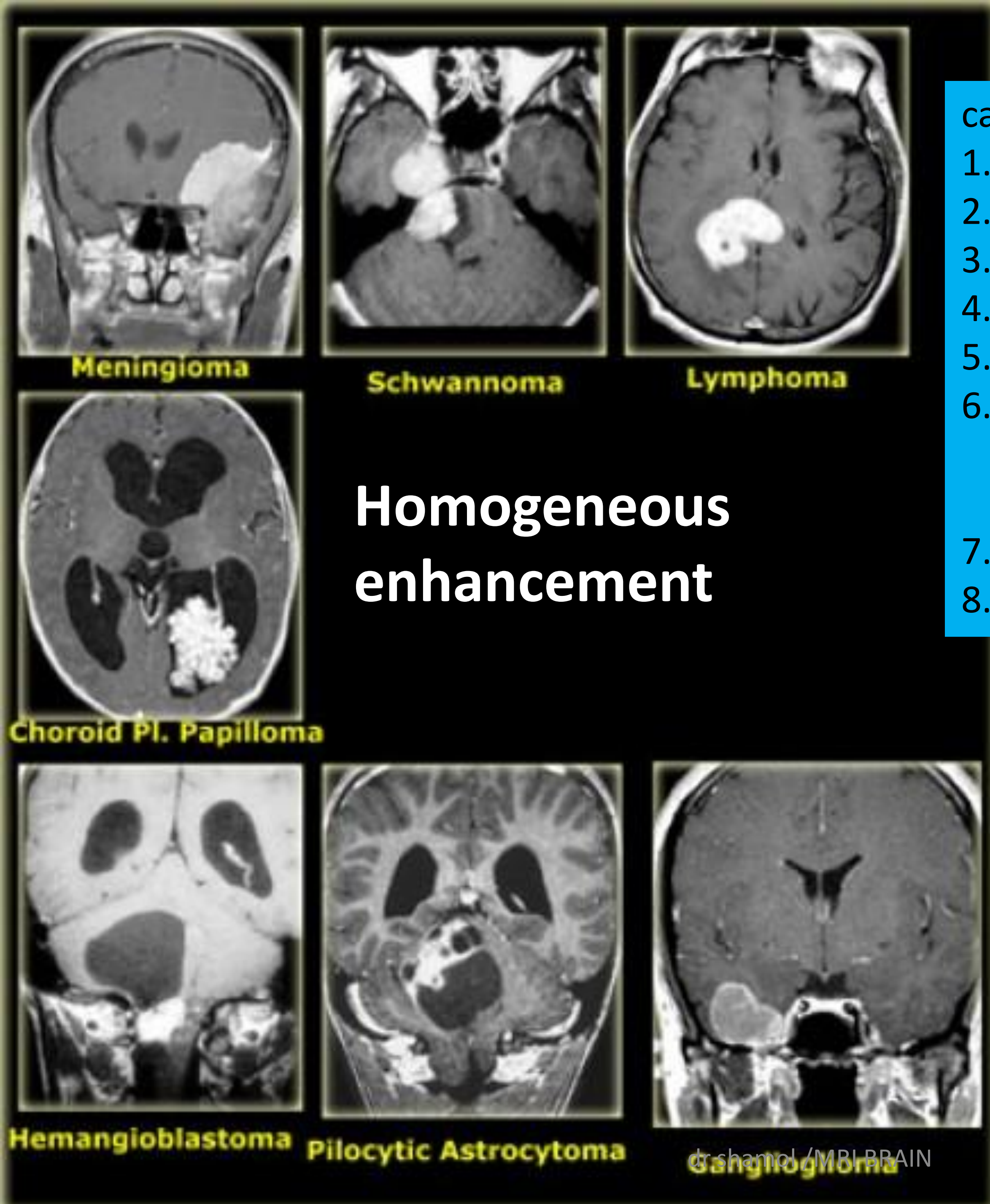
❖ Enhancement is seen when a CNS tumor destroys the BBB.

❖Extra-axial tumors such as meningiomas and schwannomas are not derived from brain cells and do not have a blood-brain arrier. Therefore they will enhance.

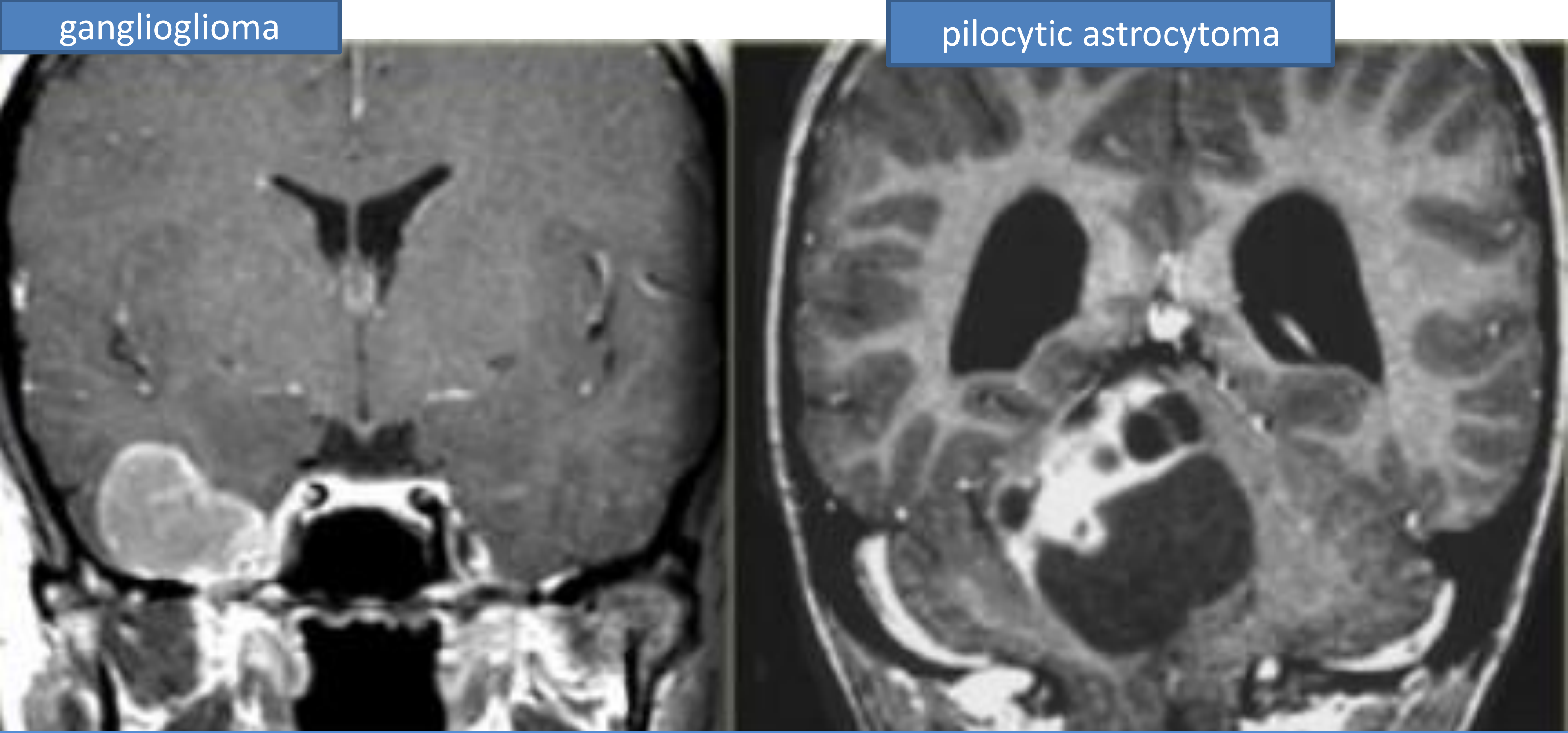
❖There is also no blood-brain barrier in the pituitary, pineal and choroid plexus regions. Therefore they will enhance

❖Some non-tumoral lesions enhance because they can also break down the BBB and may simulate a brain tumor. These lesions include like infections, abscess ,demyelinating diseases (MS) and infarctions.

Homogeneous enhancement

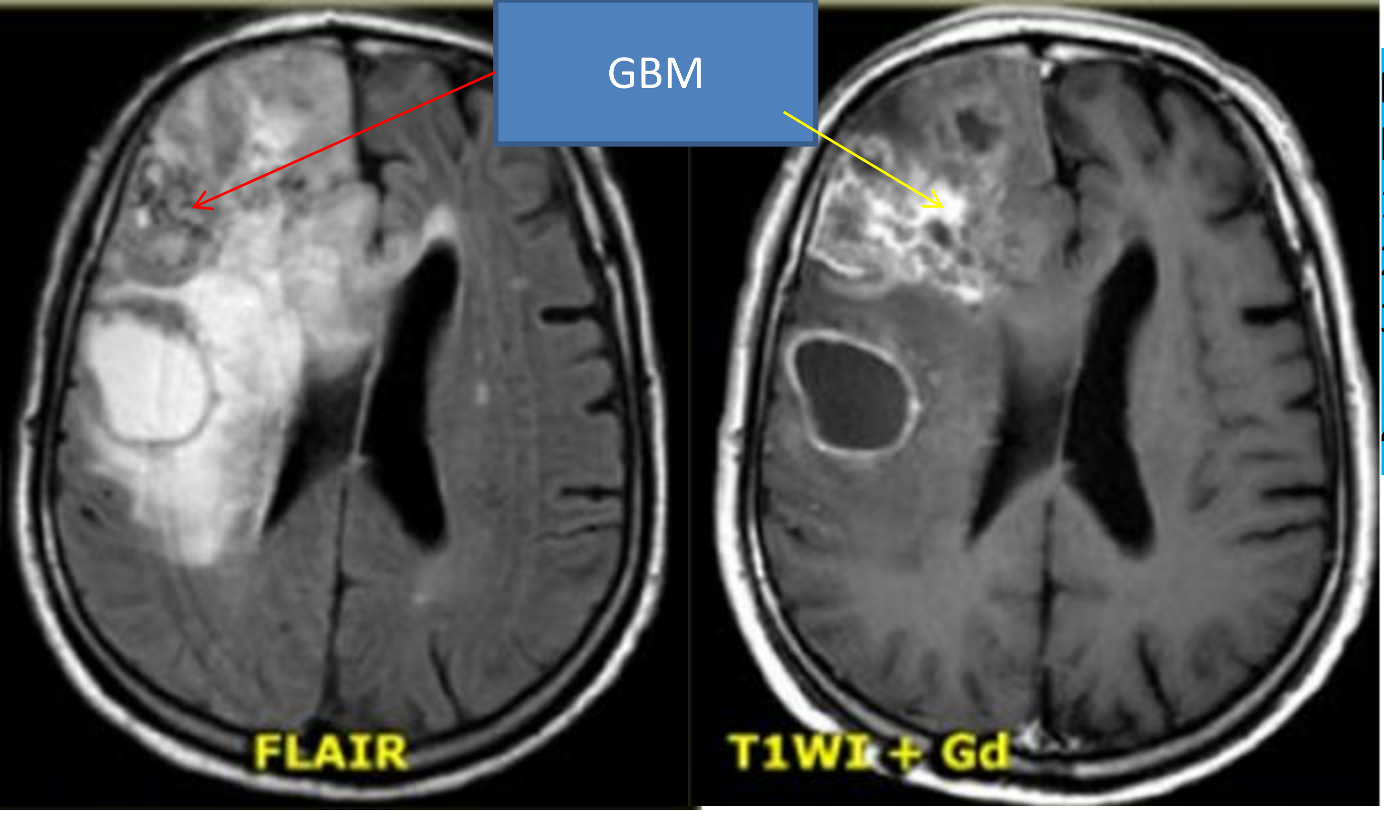


can be seen in:
1. Metastases
2. Lymphoma
3. Germinoma and
4. other pineal gland tumors
5. Pituitary macroadenoma
6. Pilocytic astrocytoma and hemangioblastoma (only the solid component)
7. Ganglioglioma
8. Meningioma and Schwannoma



low-grade glioma does not take contrast enhancement Gangliogliomas & pilocytic astrocytomas are the exceptions to this rule: they are low-grade tumors, but they enhance vividly.

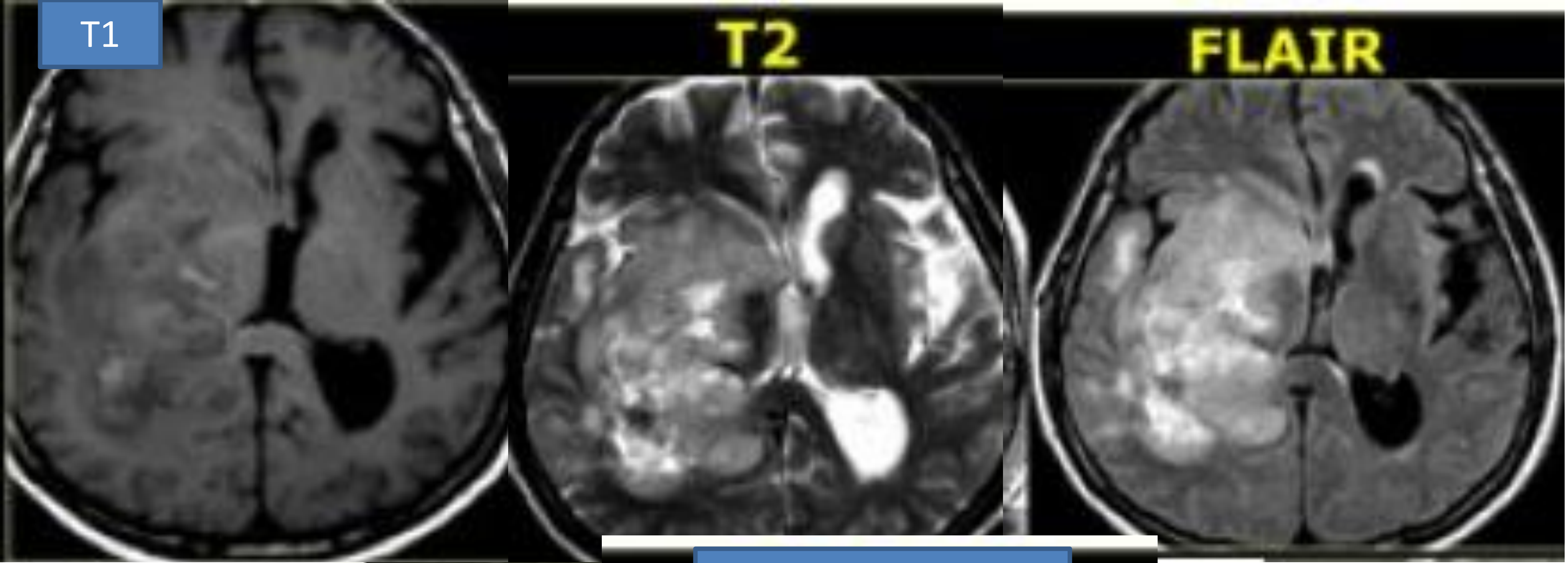
In gliomas - like astrocytomas, oligodendrogliomas and glioblastoma multiforme - enhancement usually indicates a higher degree of malignancy. Therefore when during the follow up of a the tumor starts to enhance, it is a sign of malignant transformation..

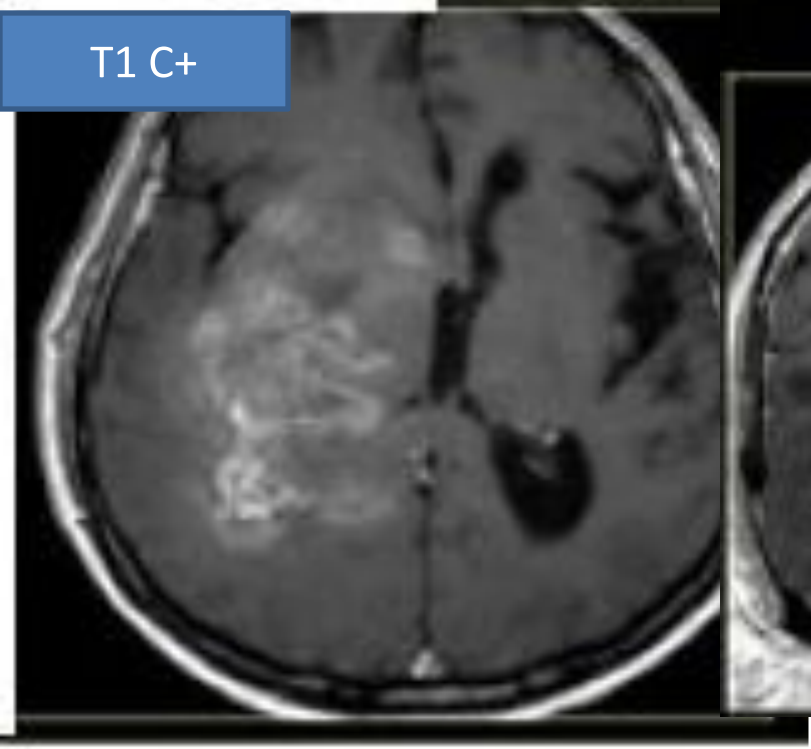


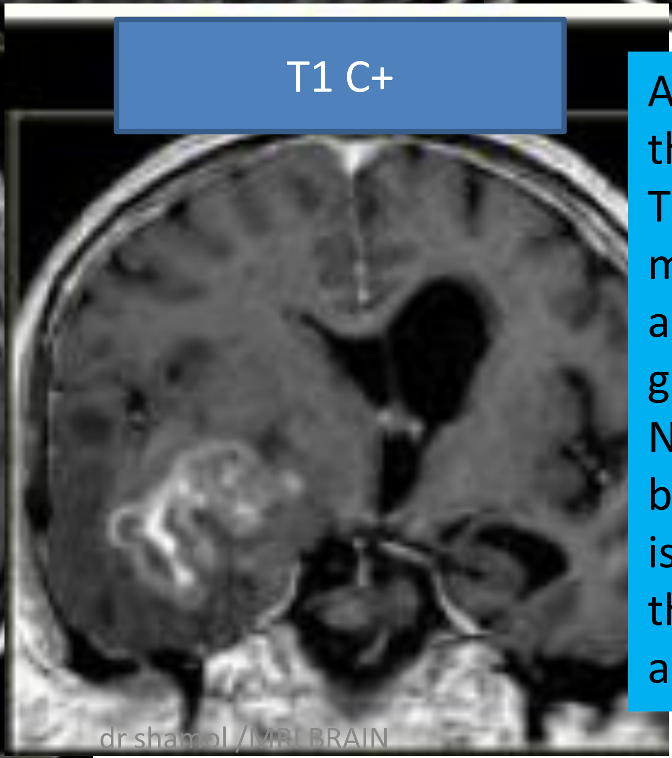
Patchy enhancement can be seen in:

1. Metastases
2. Oligodendroglioma
3. Glioblastoma multiforme
4. Radiation necrosis

glioblastoma multiforme (GBM).
The enhancement indicates that this is a high-grade tumor, but only parts of it enhance.
there is also a cystic component with ring enhancement.
The tumor cells probably extend beyond the area of edema as seen on the FLAIR image.
This is because gliomas grow infiltratively into normal brain - initially without any MR changes







Although is a large tumor, the mass-effect is limited. This indicates that there is marked infiltrative growth, a characteristic typical for gliomas.

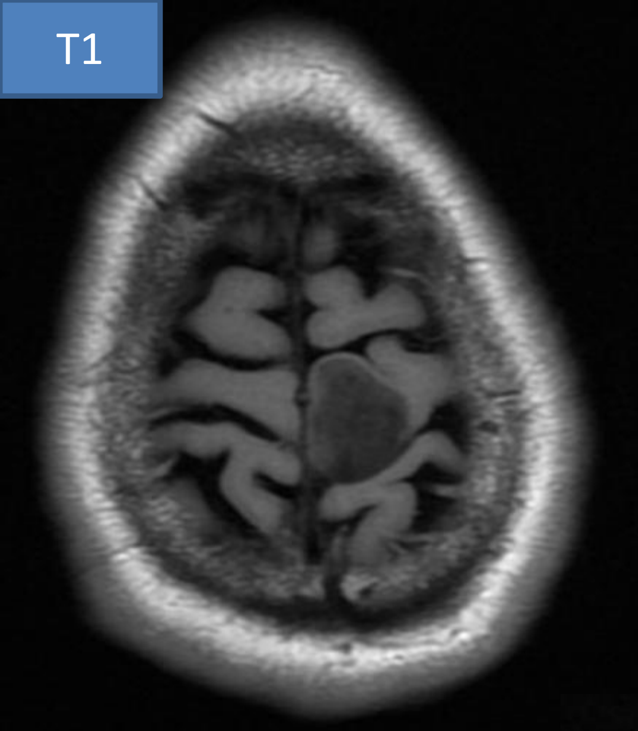
Notice the heterogeneity on both T2WI and FLAIR. There is patchy enhancement. All these findings are typical for a GBM

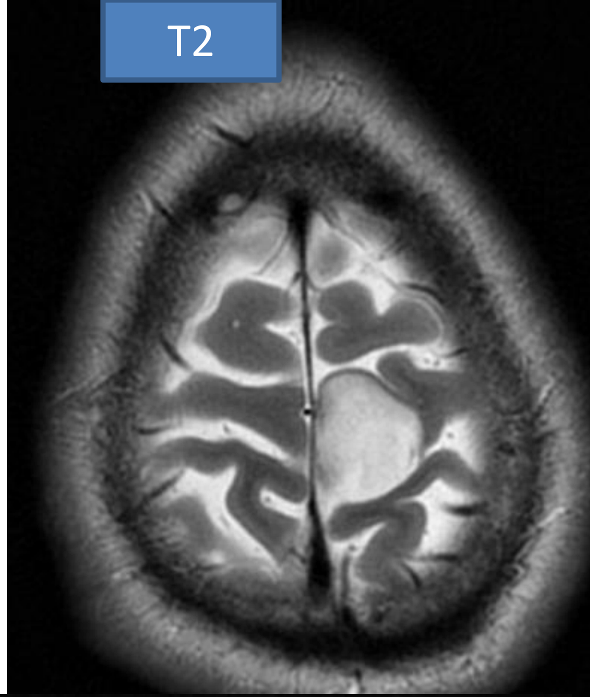
No enhancement is seen in:

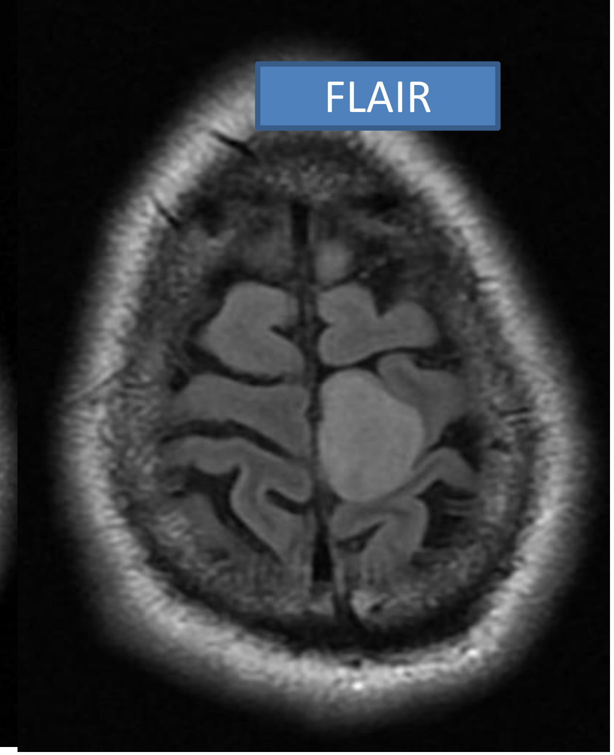
Low grade astrocytomas

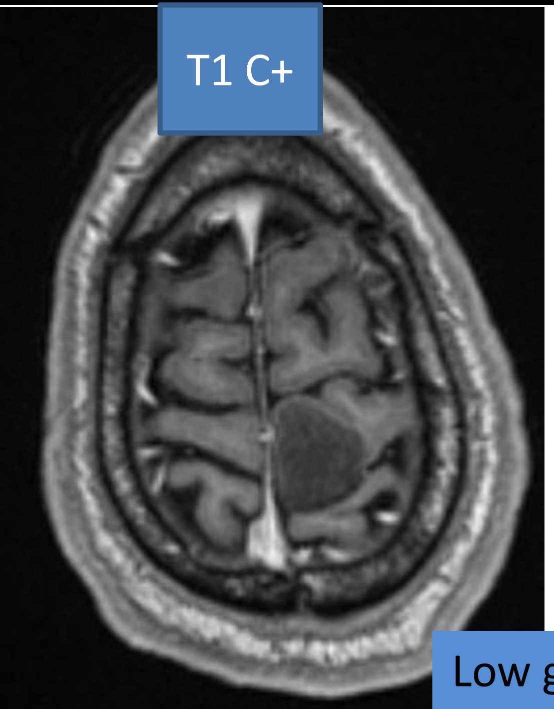
Cystic non-tumoral lesions:

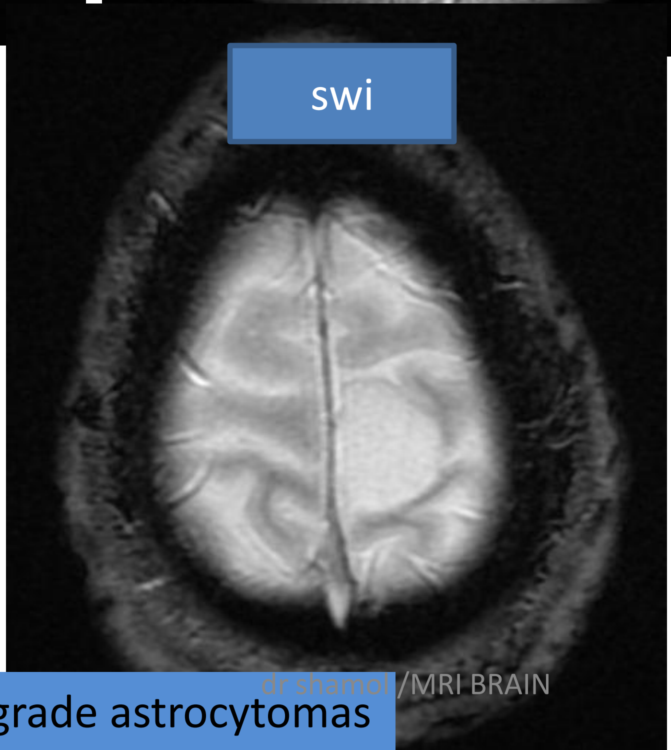
- Dermoid cyst
- Epidermoid cyst
- Arachnoid cyst











There is an abnormal poorly enhancing well defined intra-axial lesion in the subcortical white matter of left high parietal lobe. The lesion is seen as hypointense on T1W, hyperintense on T2W and FLAIR, showing no blooming on GRE or restriction on DWI

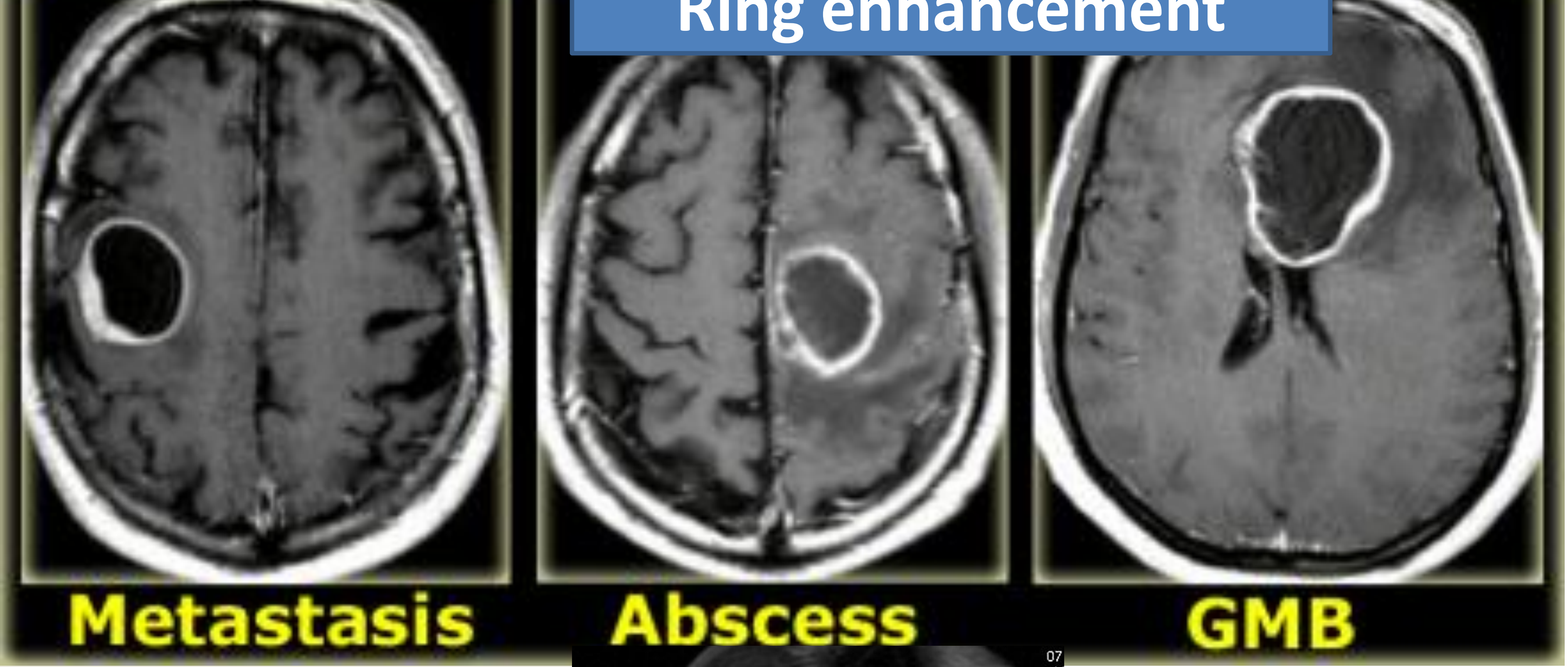
Low grade astrocytomas

Ring enhancing lesions

Ring enhancing cerebral lesions includes:

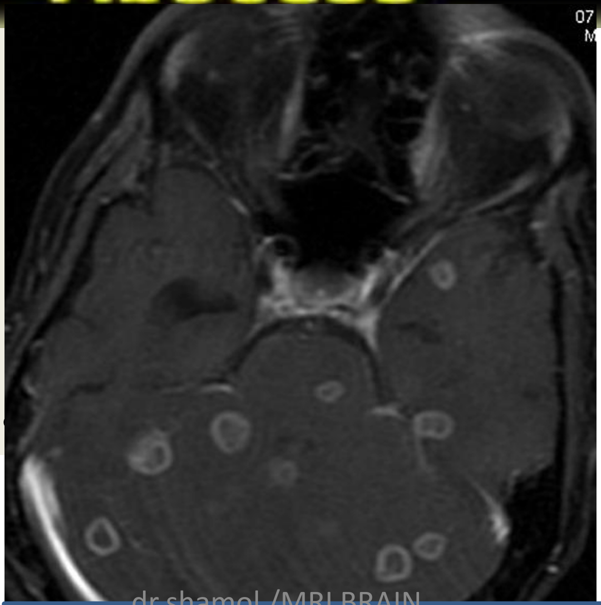
1. **cerebral abscess**
2. **tuberculoma**
3. **metastasis**
4. neurocysticercosis
5. glioblastoma
6. subacute infarct /haemorrhage /contusion
7. demyelination (incomplete ring)
8. radiation necrosis
9. lymphoma - in an immunocompromised patient

Ring enhancement



Ring enhancement is seen in

1. abscesses
2. metastases
3. Tuberculoma
4. high-grade gliomas.
5. some MS-plaques and
6. sometimes in an old hematom



Tuberculoma

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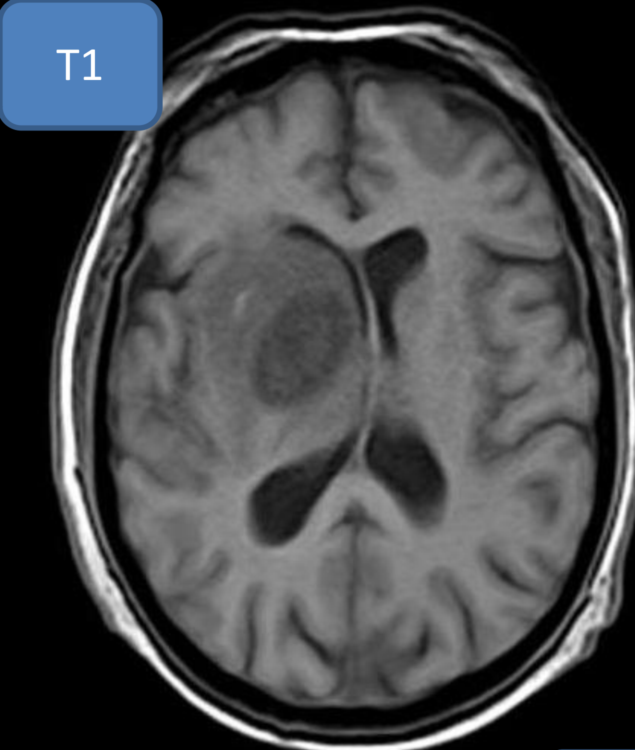
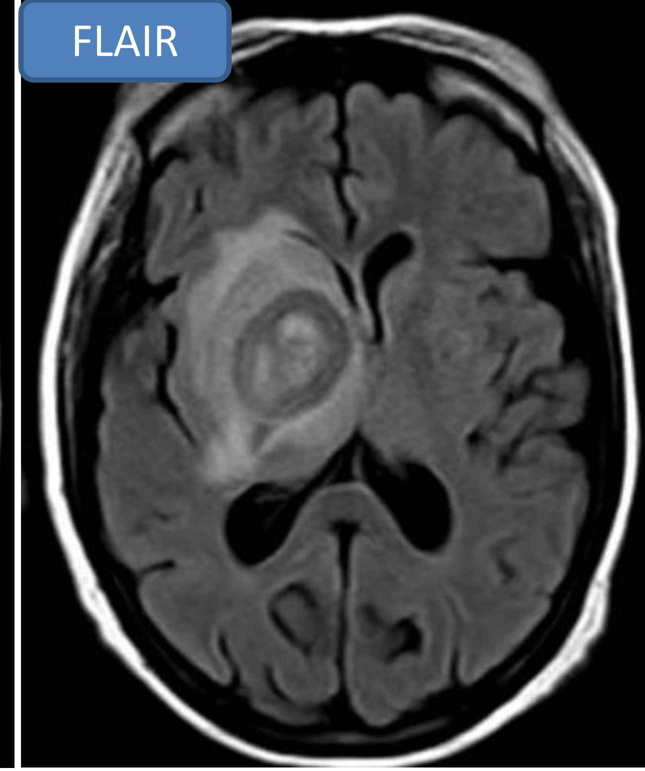
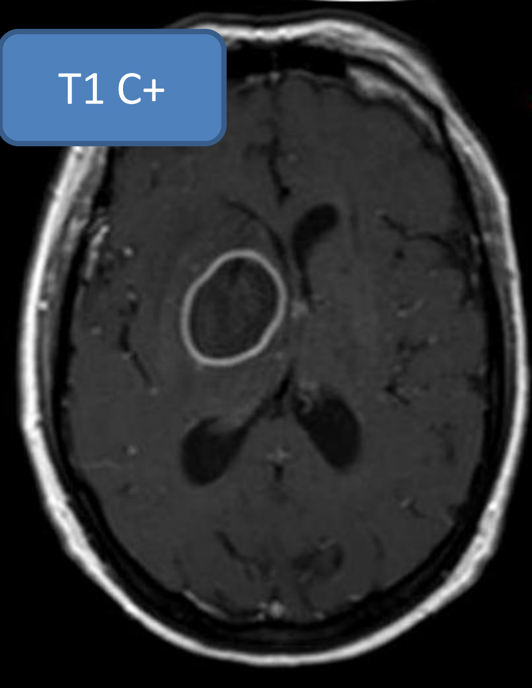
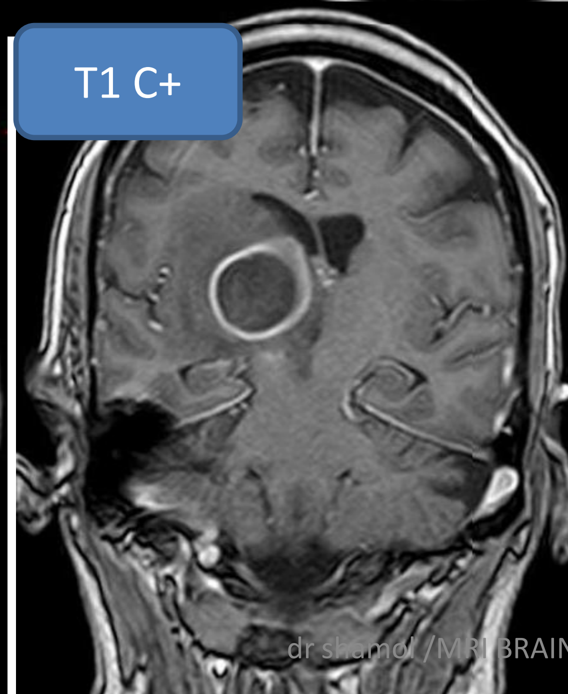
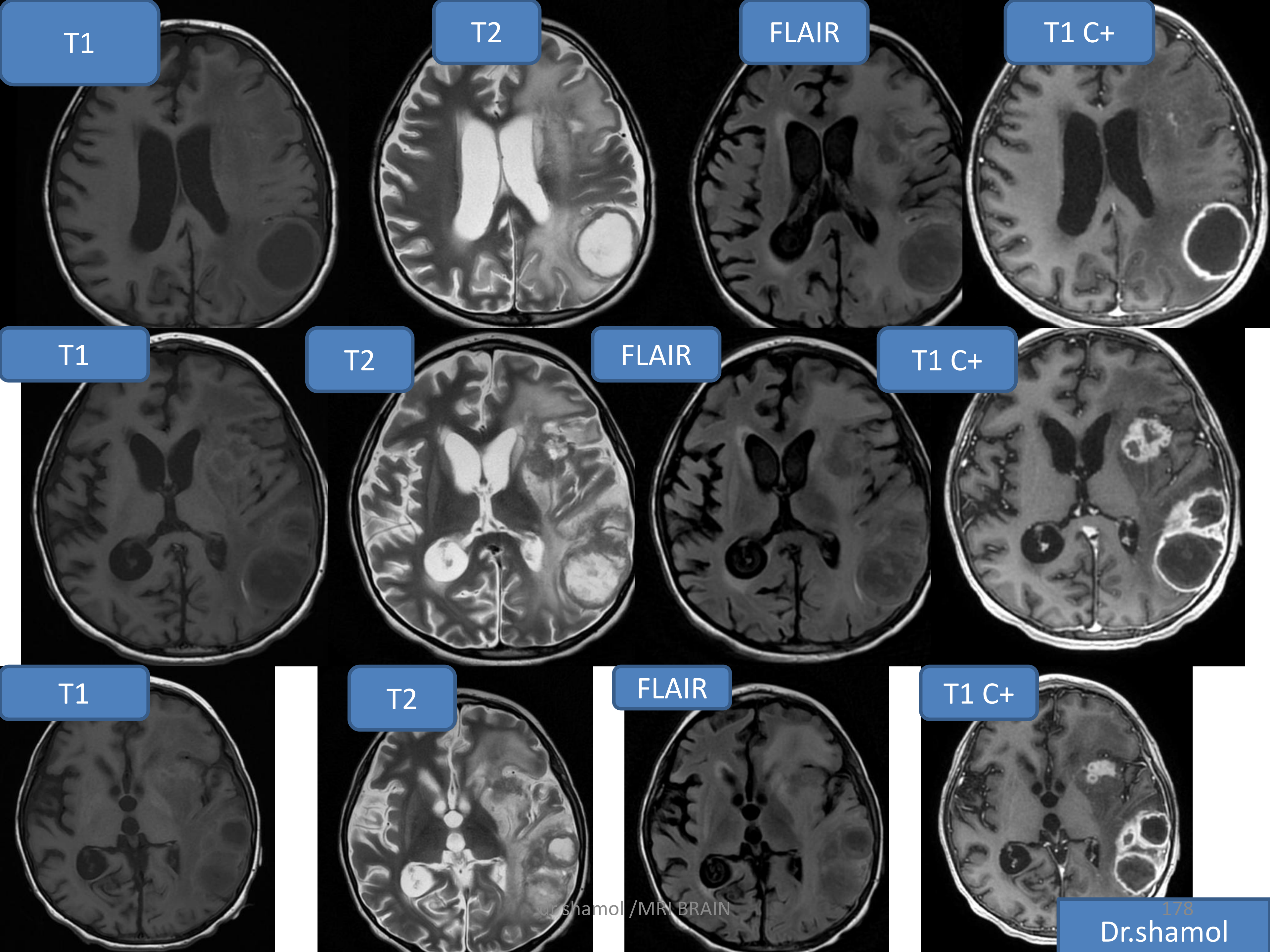


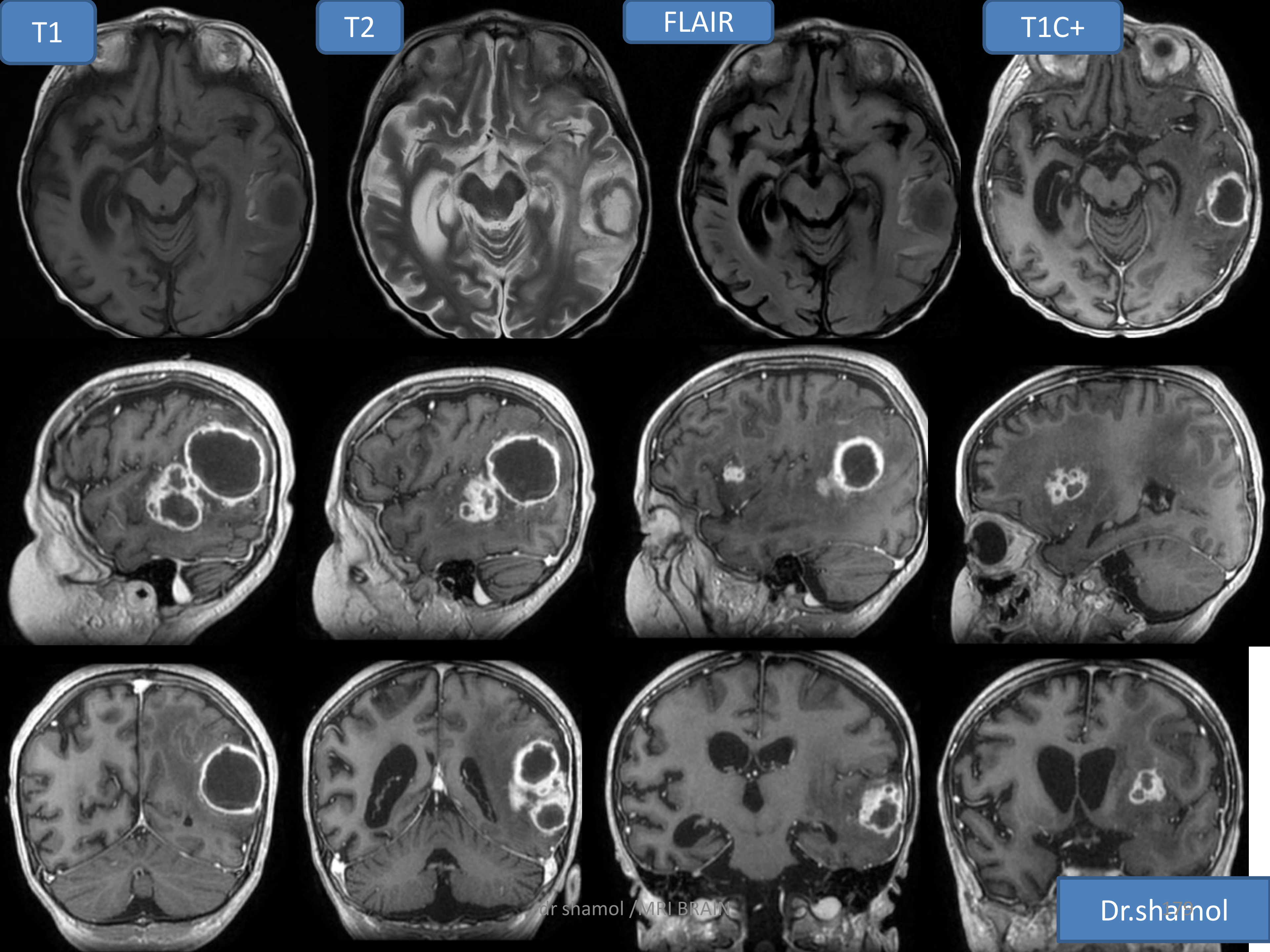






Headache followed by disturbed conscious level. The patient was previously diagnosed with bacterial endocarditis
Right basal ganglionic well defined cystic lesion that exhibits low signal of its content in T1 and FLAIR and high signal in T2. Its wall displays low signal in T2 and FLAIR while hyperintense signal in T1 likely from free radicals and enhances avidly following contrast injection (ring enhancement). It is surrounded by perifocal edema and exerts positive mass effect upon the adjacent lateral ventricle.
Diagnosis: Intracerebral absces


T1
T2
FLAIR
T1 C+
T1
T2
FLAIR
T1 C+
T1
T2
FLAIR
T1 C+
Dr.shamol


T1
T2
FLAIR
T1C+
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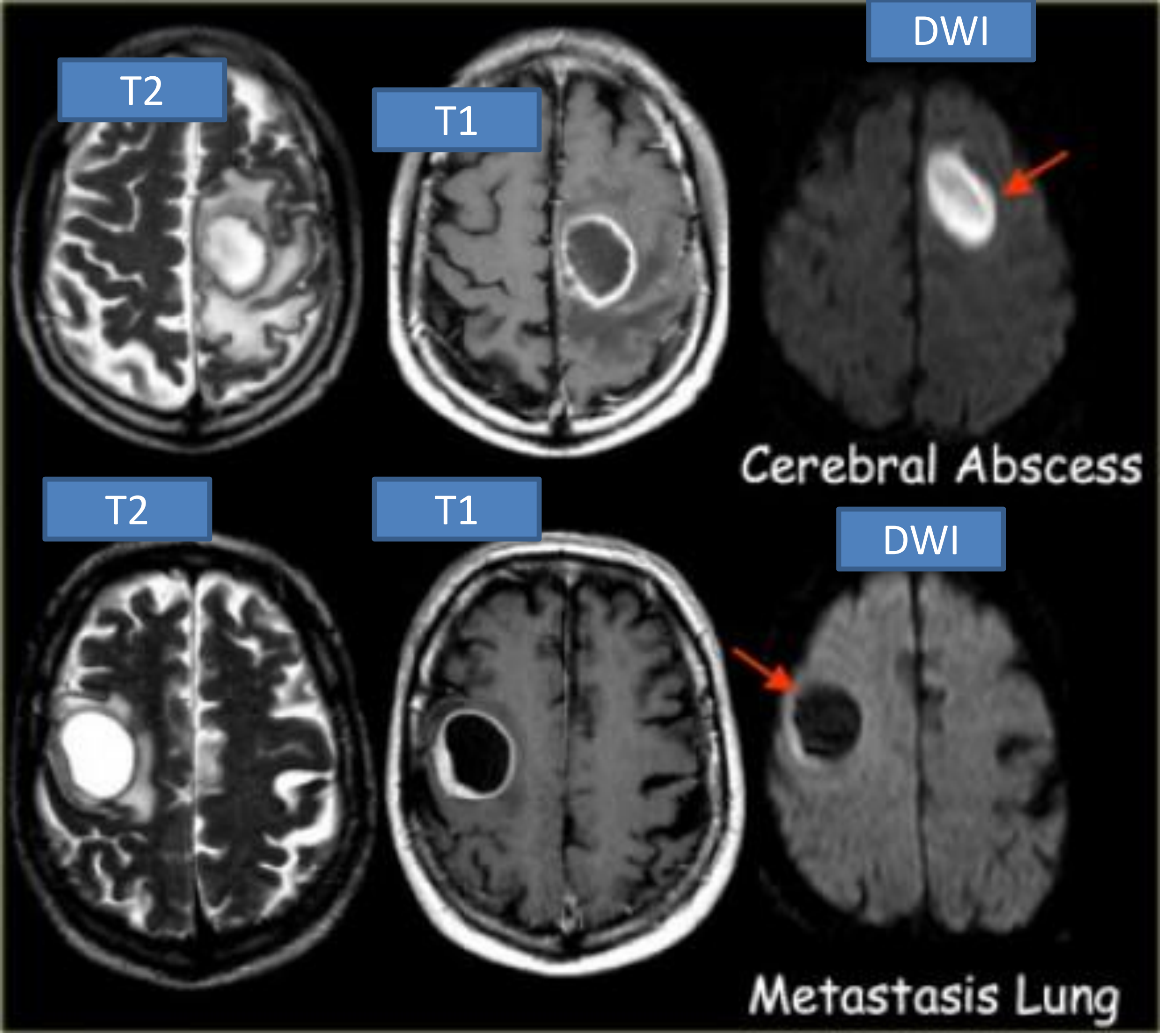
Ring Enhancing wall characteristics	thick and nodular favours	neoplasm
	thin and regular favours	abscess
	incomplete ring opened toward the cortex	Demyelination
surrounding oedema	extensive oedema relative to lesion size	favours abscess
	increased perfusion	favours neoplasm
central fluid	restricted diffusion favours	Abscess
	absence of diffusion restriction favour	tumour
	absence of diffusion restriction with a central necrotic component	Metastases
number of lesions	similar sized rounded lesions at grey-white matter junction	Favors metastases or abscesses
	irregular mass with adjacent secondary lesions embedded in the same region of 'oedema'	Favours GBM

HOW WILL DIFFERNTIATE ABSCESS AND TUMOUR

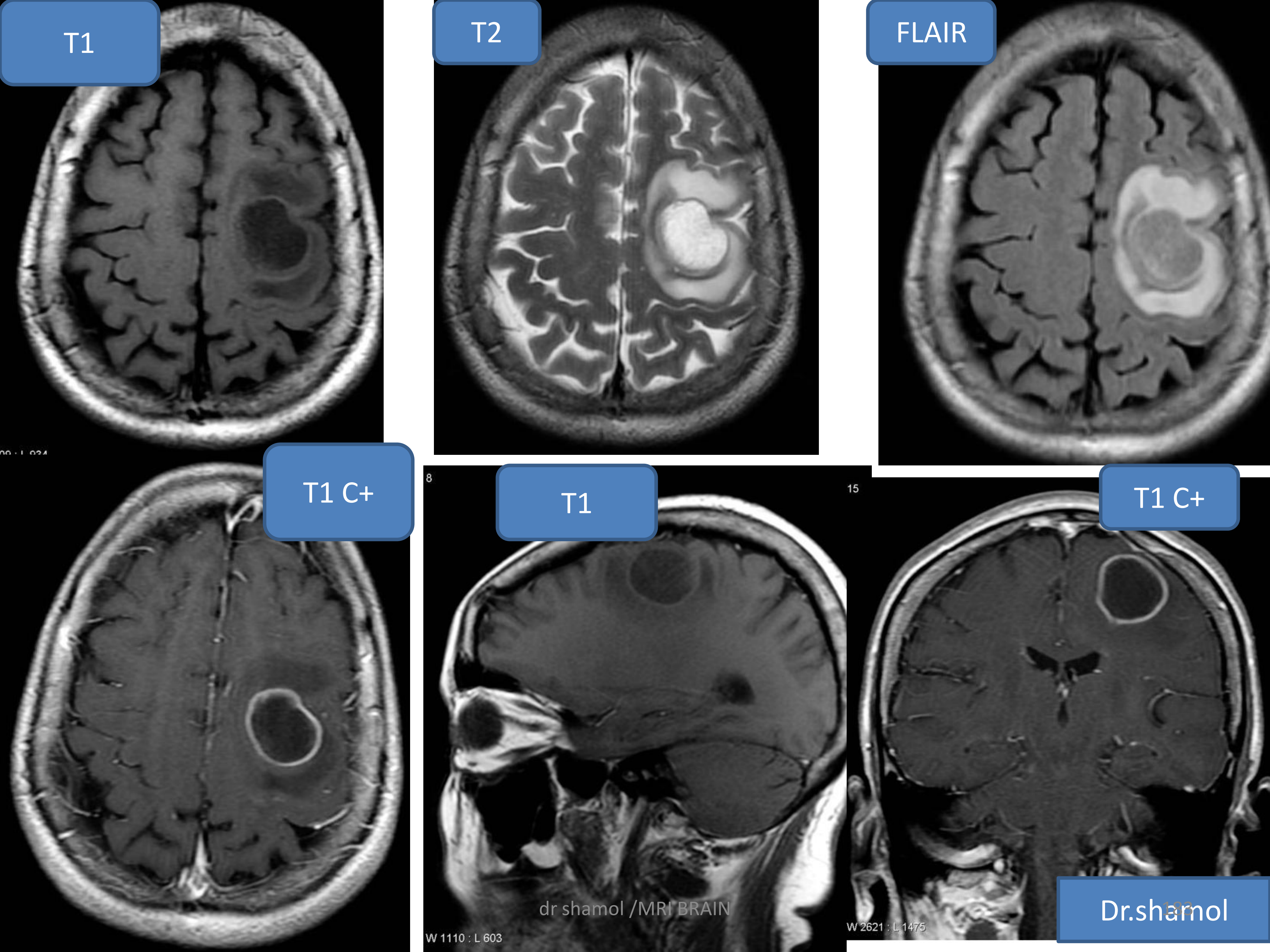


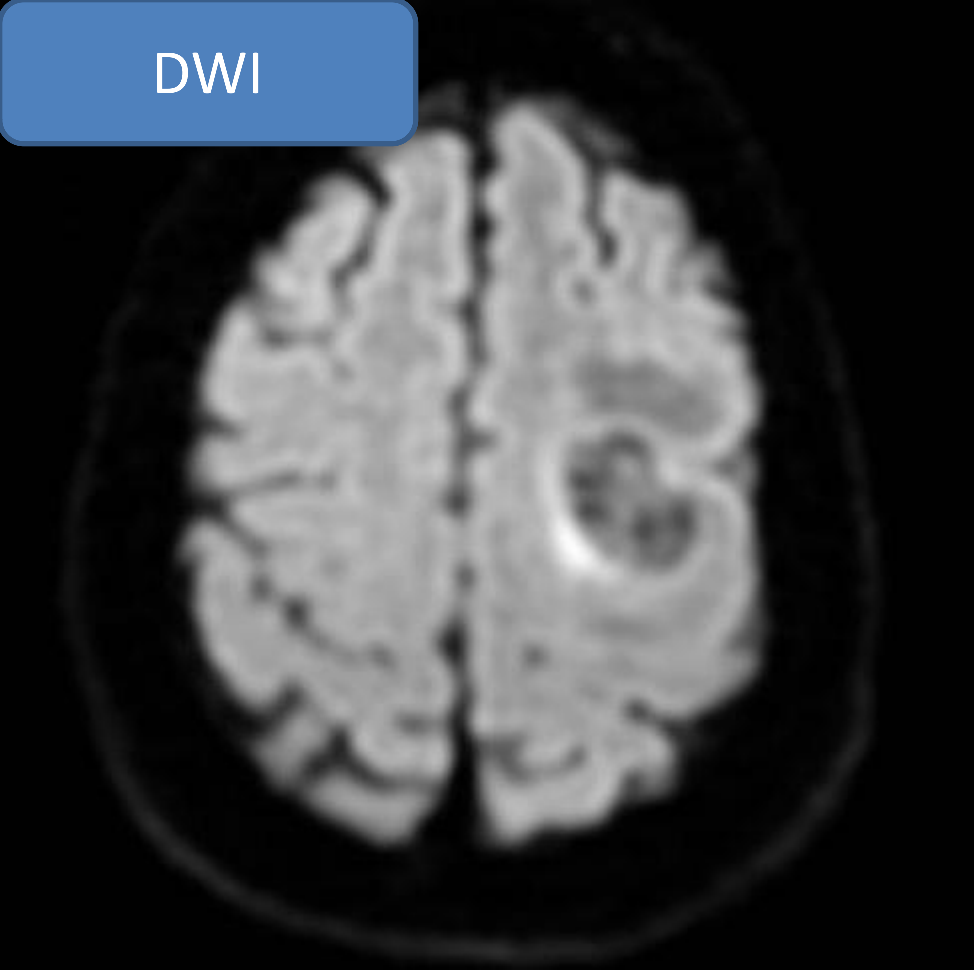
Diffusion weighted imaging

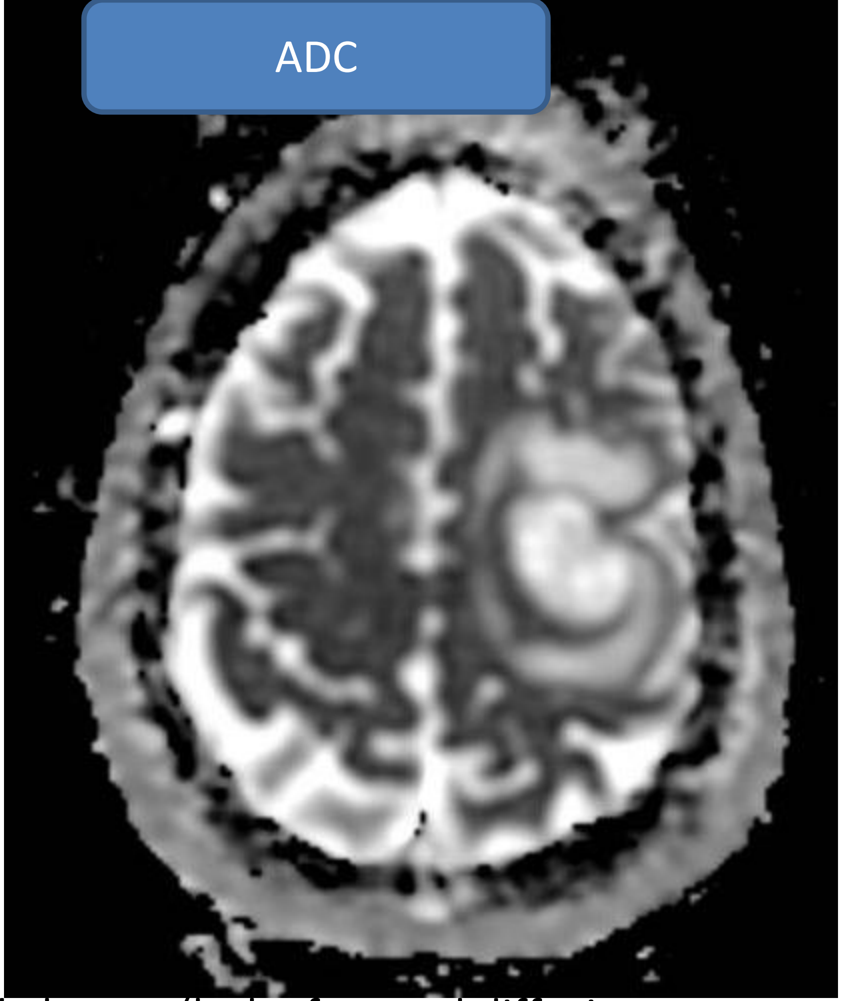
- Normally water protons have the ability to diffuse extracellularly and loose signal.
- High intensity on DWI indicates restriction of the ability of water protons to diffuse extracellularly.
- Restricted diffusion is seen in abscesses, epidermoid cysts and acute infarction (due to cytotoxic edema).
- In cerebral abscesses the diffusion is probably restricted due to the viscosity of pus, resulting in a high signal on DWI.

❖In most tumors there is no restricted diffusion - even in necrotic or cystic components.

❖This results in a normal, low signal on DWI.


T1
T2
FLAIR
T1 C+
8
T1
15
T1 C+
dr shamol /MRI BRAIN
W 1110 : L 603
W 2621 : L 1475
Dr.shamol





Perfect' ring enhancing lesion which is not a cerebral abscess (lack of central diffusion restriction on DWI and relative paucity of oedema)

Ring-enhancing mass in the left posterior frontal lobe with a small amount of surrounding vasogenic oedema. Some peripheral (but not central) diffusion restriction.

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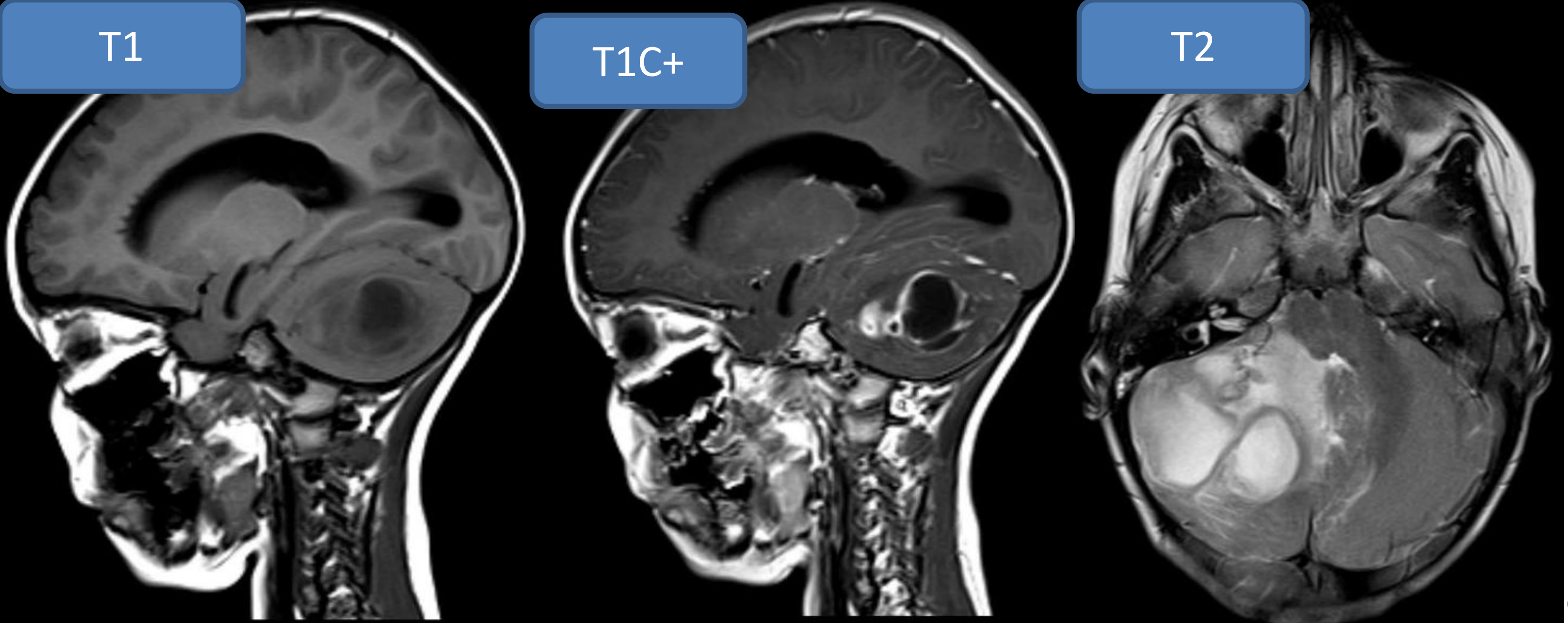

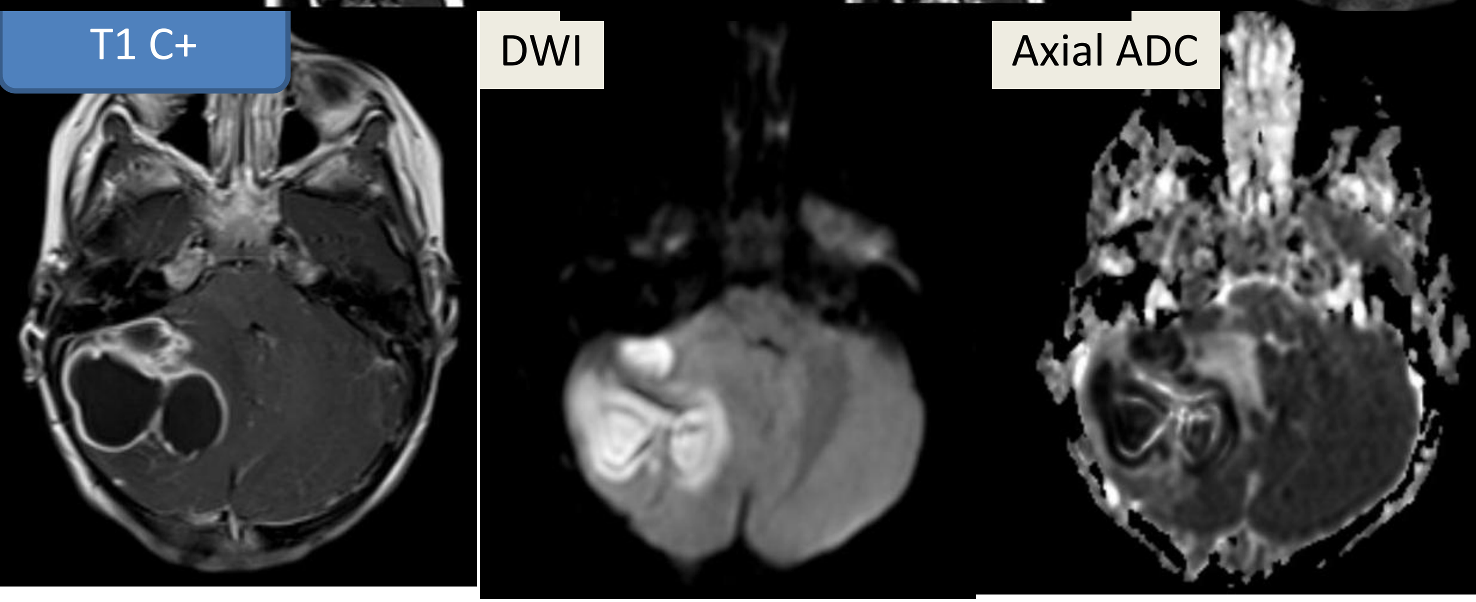

ent went on to have a craniotomy and aspiration of the content of the lesion.

Gram stain: ++++

Gram positive cocci in chains and pairs, confirmed to be Strep pneumoniae.

Selected images from an MRI of the brain demonstrates a large peripherally enhancing mass in the right cerebellar hemisphere. It demonstrates prominent central restricted diff[illegible] surrounded by oedema and results in mass effect and compression of the 4th ver

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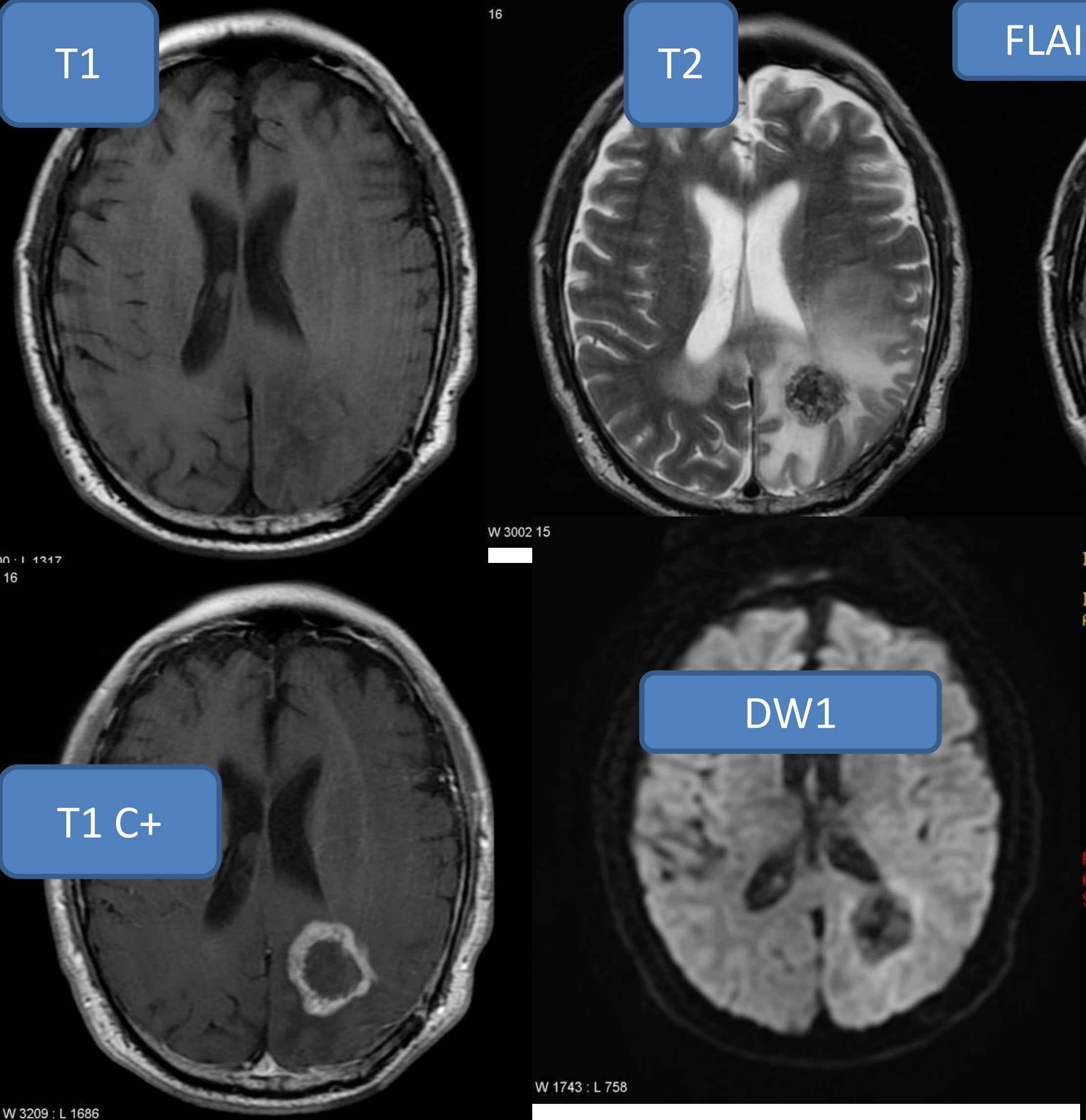
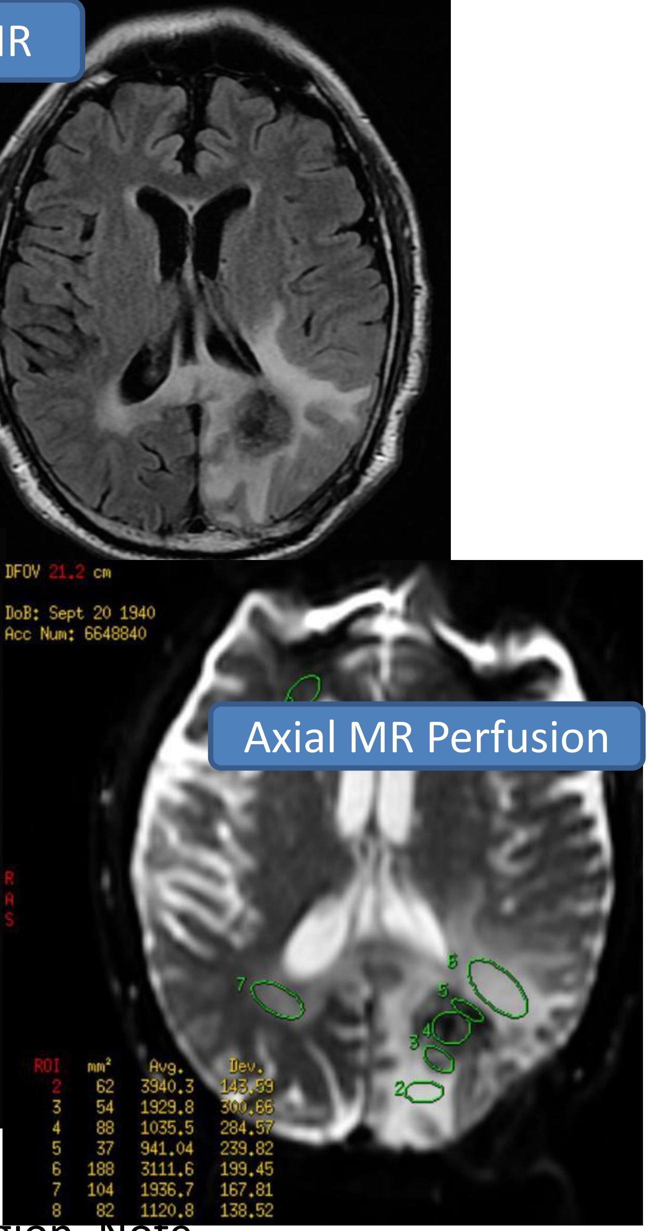
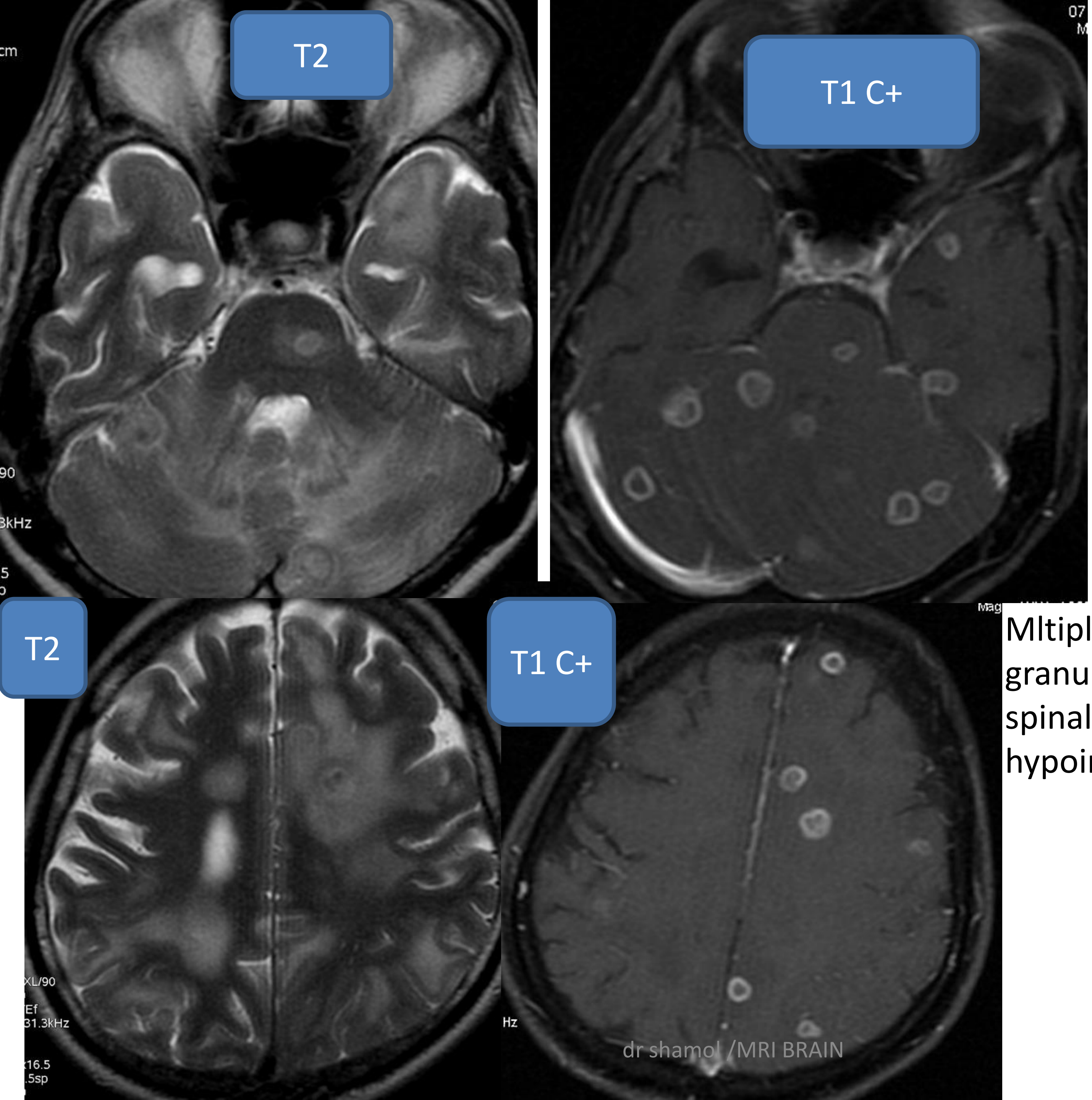




Cerebral radiation necrosis

Ring-enhancing lesion in the left parieto-occipital region. Note the reduced perfusion in the enhancing component helping to differentiate it from recurrence



Mltiple ring enhancing granulomas in the brain and spinal cord. These are hypointense on T2WI.

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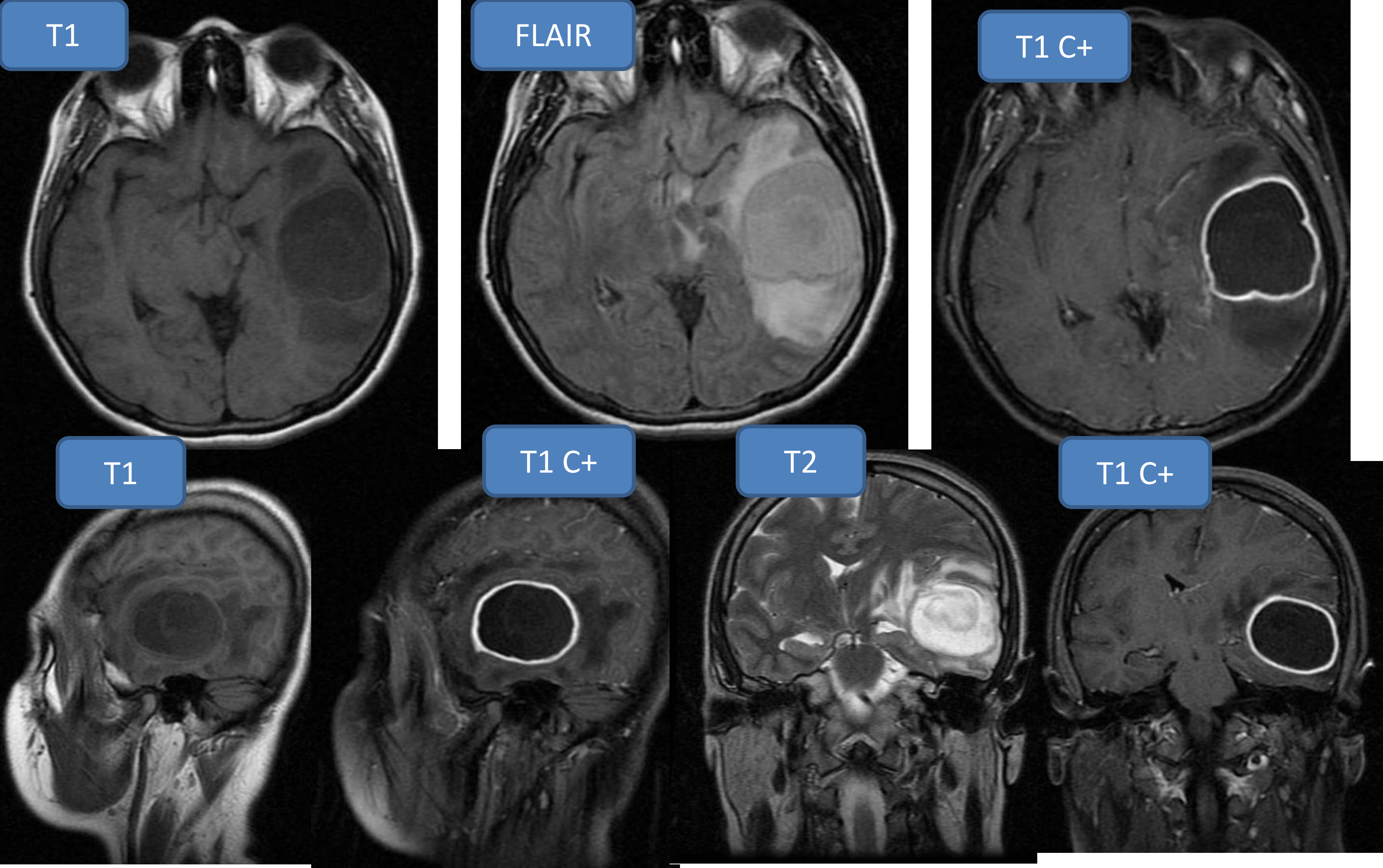

Found intra-axial lesion with vasogenic edema that have a hyper intense thin-wall in T1 and hypo intense wall at T2. Post gadolinium shows rim-enhancement.

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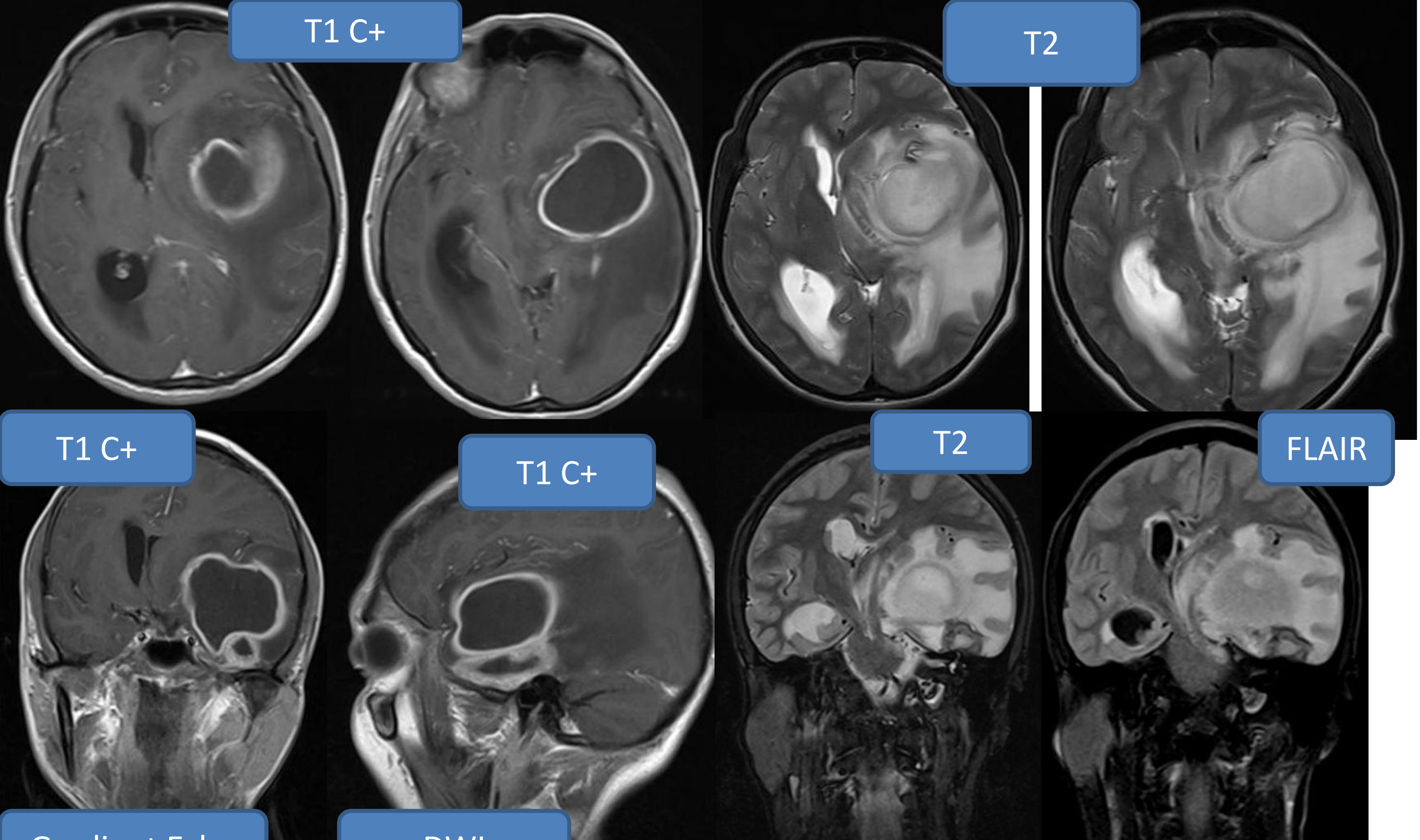
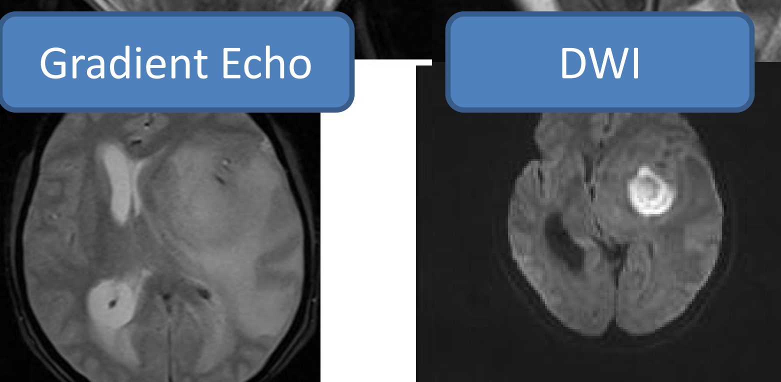
Case Discussion

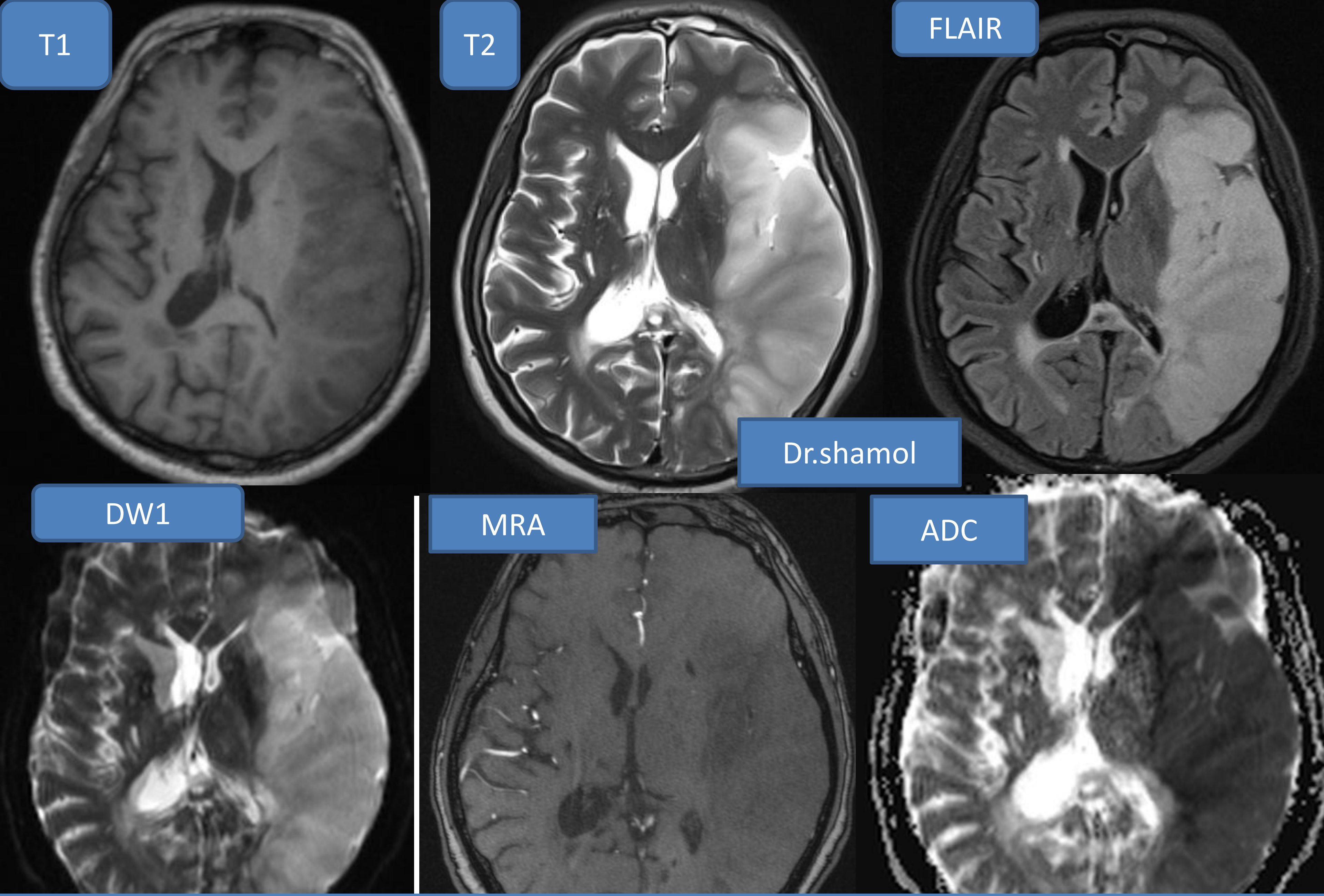
The CT and MRI findings are compatible with pyogenic brain abscess in late capsular stage.



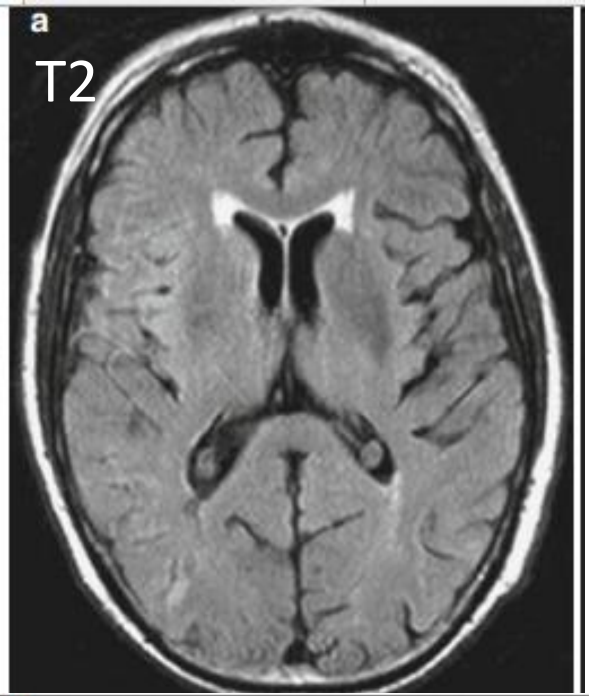


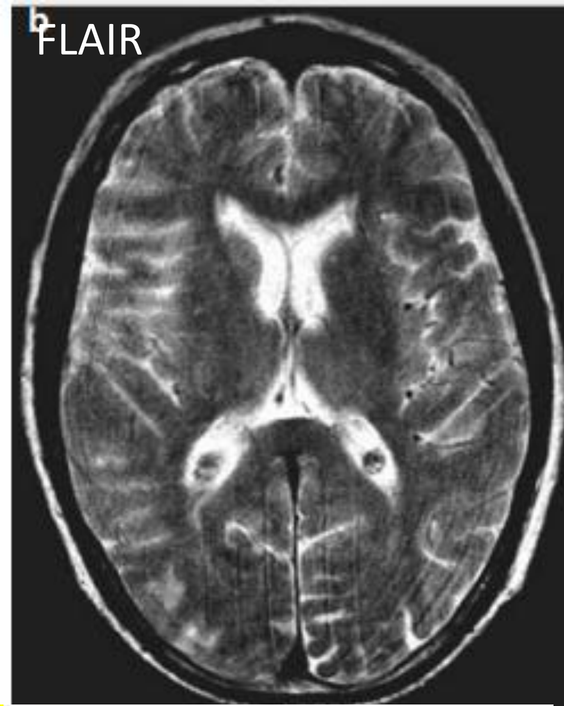
Known case of sickle cell anemia. Complaining of fever and right side weakness. Large well defined thick wall enhancing lesion is seen at the left temporal region, which shows central area of restricted diffusion on DWI. Another smaller lesion is seen near to the above mentioned lesion. Moderate perilesional edema present. Adjacent meningeal enhancement noted. Mass effect with midline shift of 13 mm to the right side with compression and effacement over the left lateral ventricle and basal cistern.

INFARCTION IN MRI



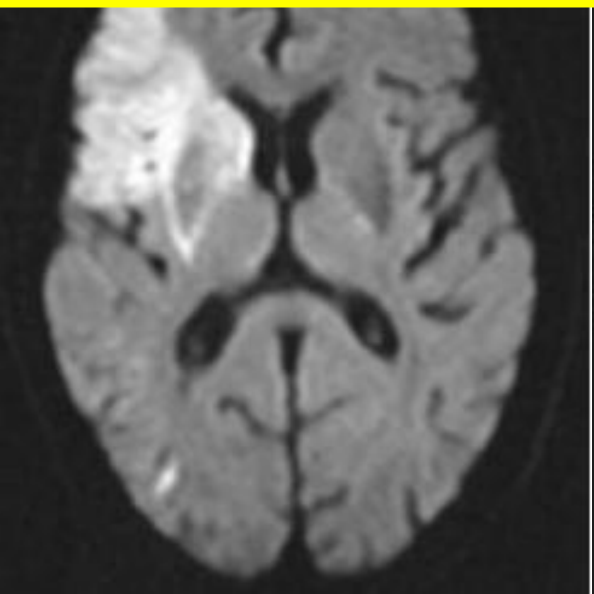
Large T2/Flair hyperintensity area with restricted diffusion in the left middle cerebral artery territory.MRA reveals no signal in the left middle cerebral artery, from M2 segment onward.

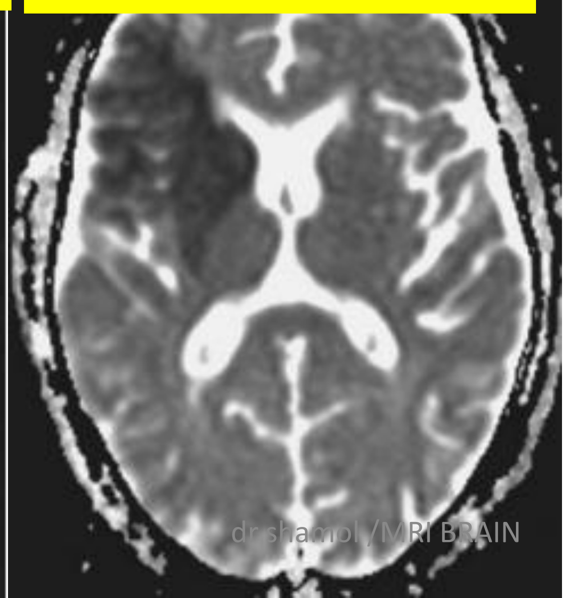




Axial diffusion-weighted image (DWI),

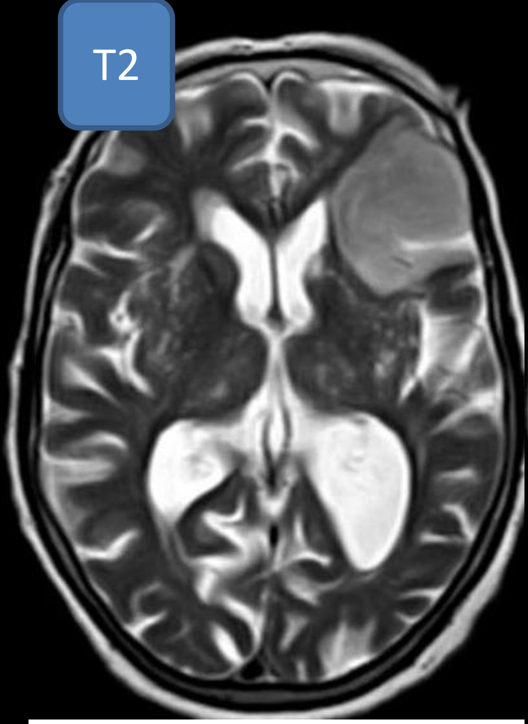
Axial apparent diffusion coeffi cient map (ADC)

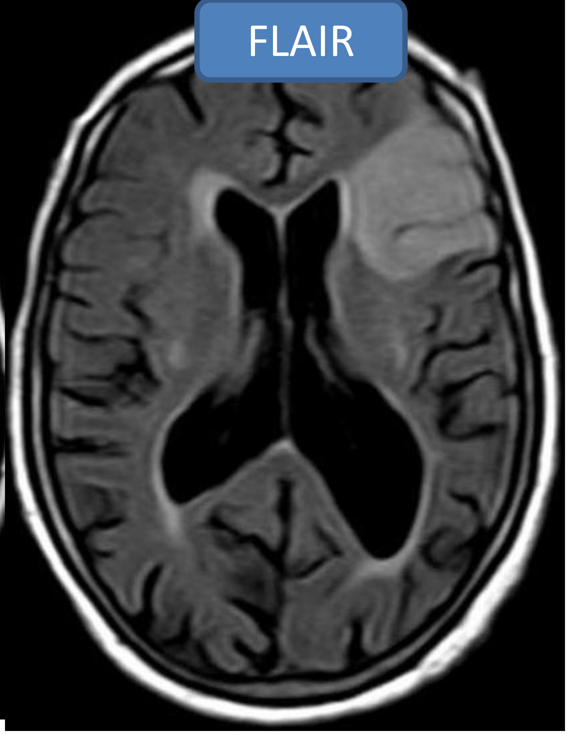


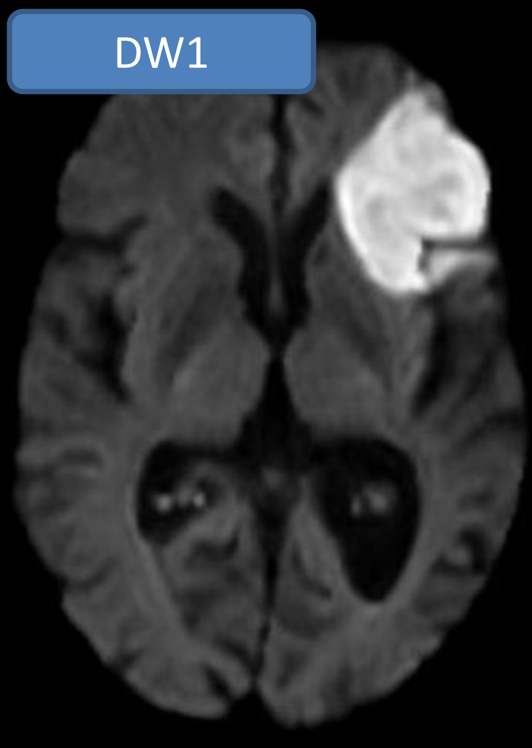


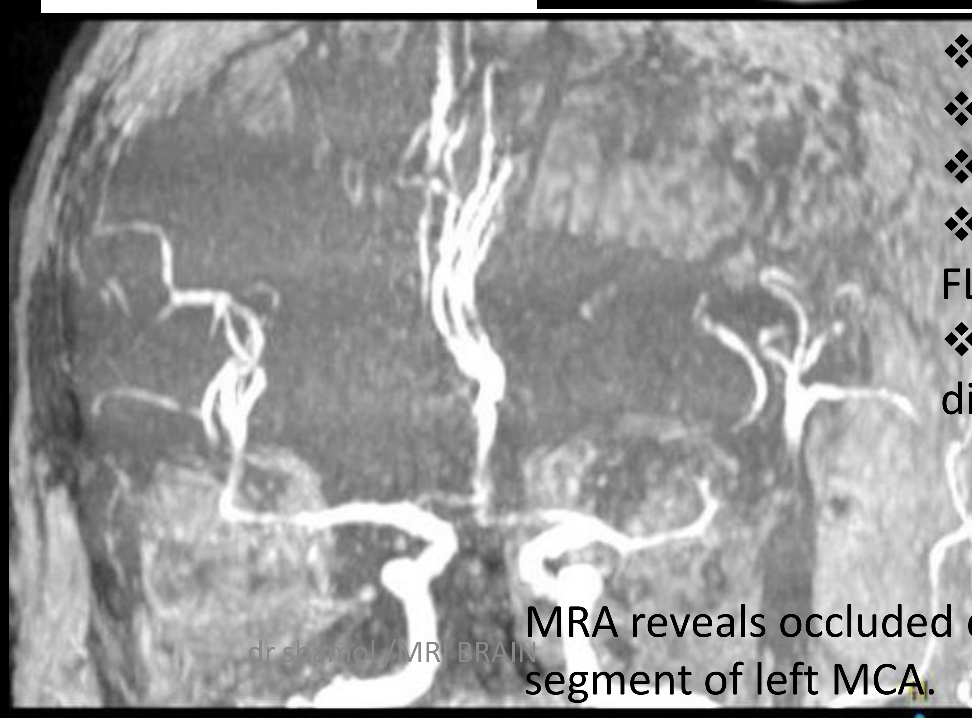
STROKE after 2 hrs

The axial FLAIR and TSE T2-WI are within normal limits. Image quality is somewhat degraded by motion artifacts. The DWI shows a focal area of bright signal intensity in the right operculofrontal and sylvian region. On the ADC map, the area of restricted diffusion is confi rmed as a hypointense lesion (decreased ADC). Signal abnormalities in the DWI and ADC maps indicate cytotoxic edema. The fi ndings are consistent with hyperacute infarction of the right middle cerebral artery









- ❖infarction displays,
- ❖ low signal in T1
- ❖ bright signal in T2
- ❖bright signal in FLAIR
- ❖restricted diffusion

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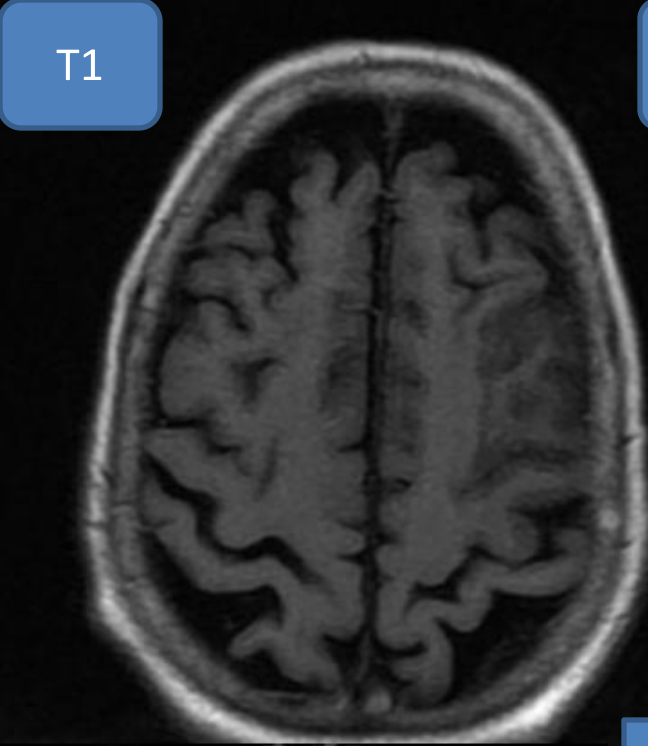
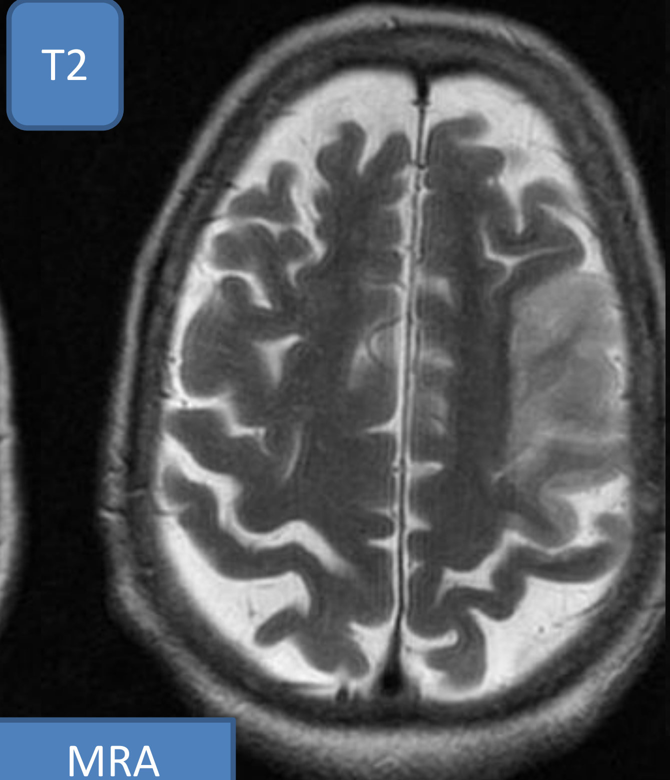
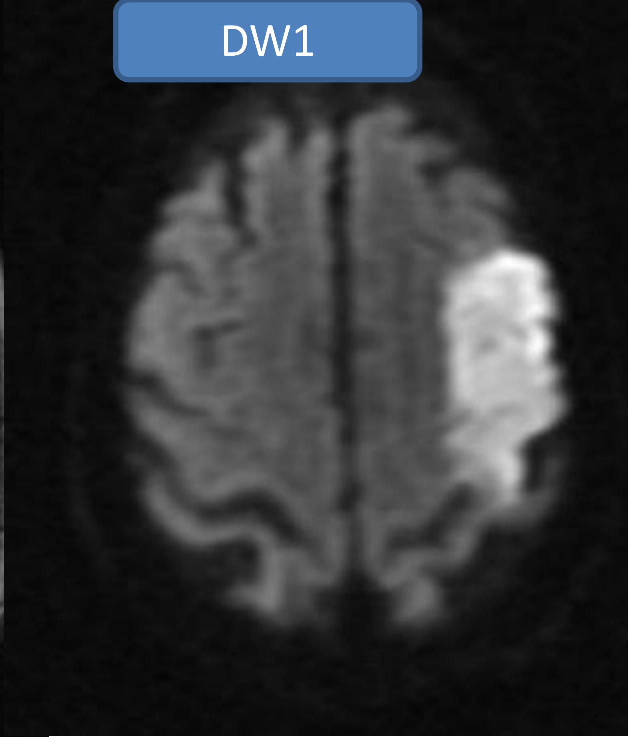
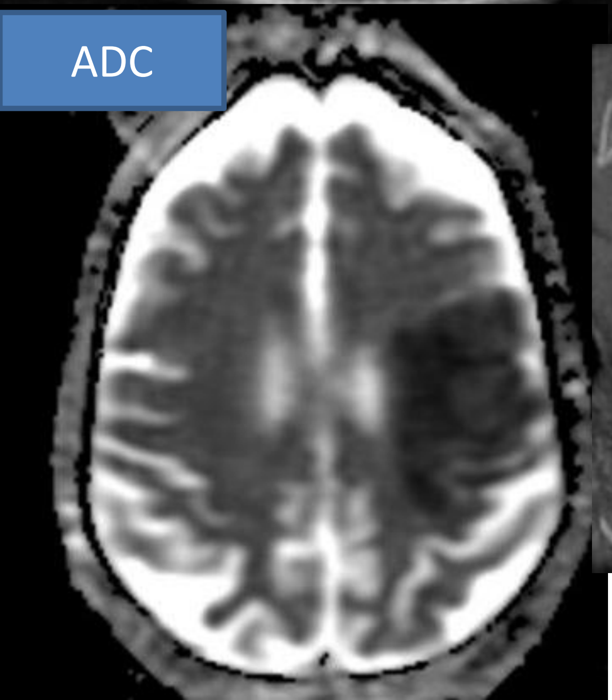
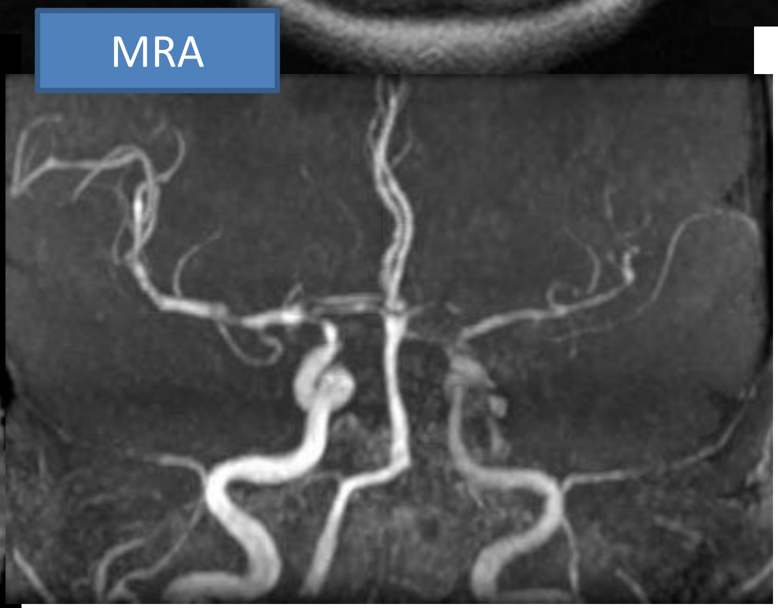
MRA reveals occluded distal part of M2 segment of left MCA.



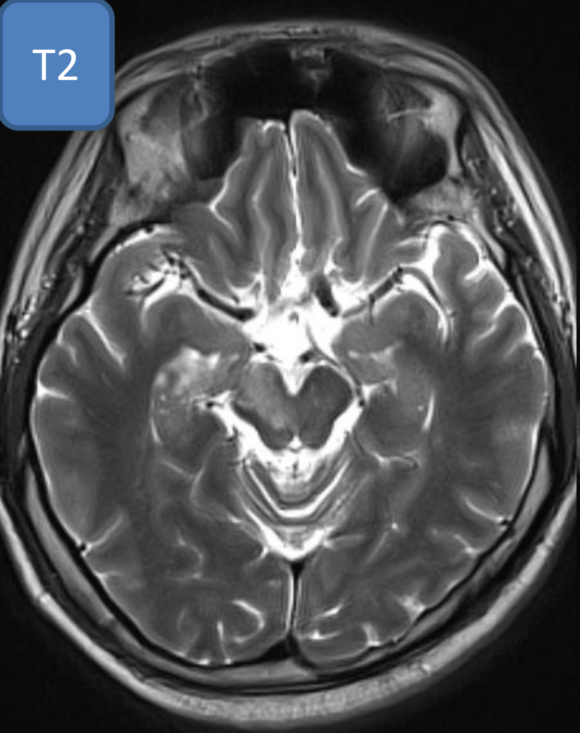


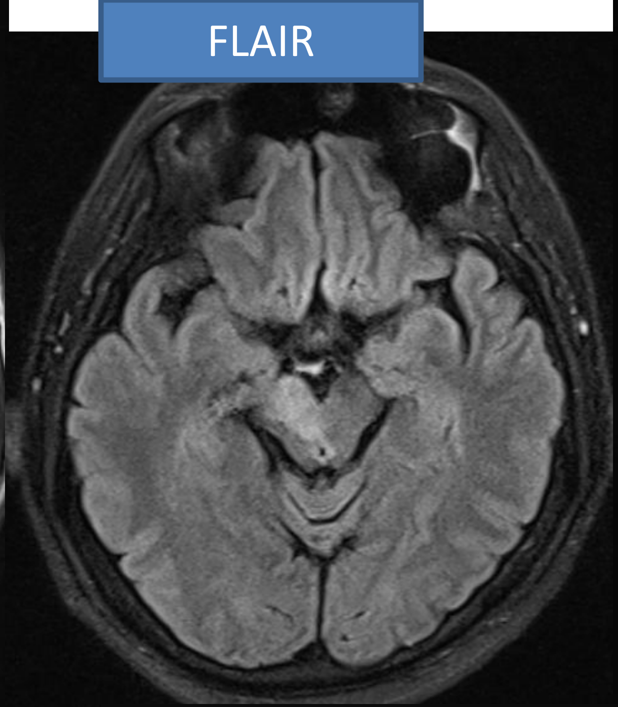


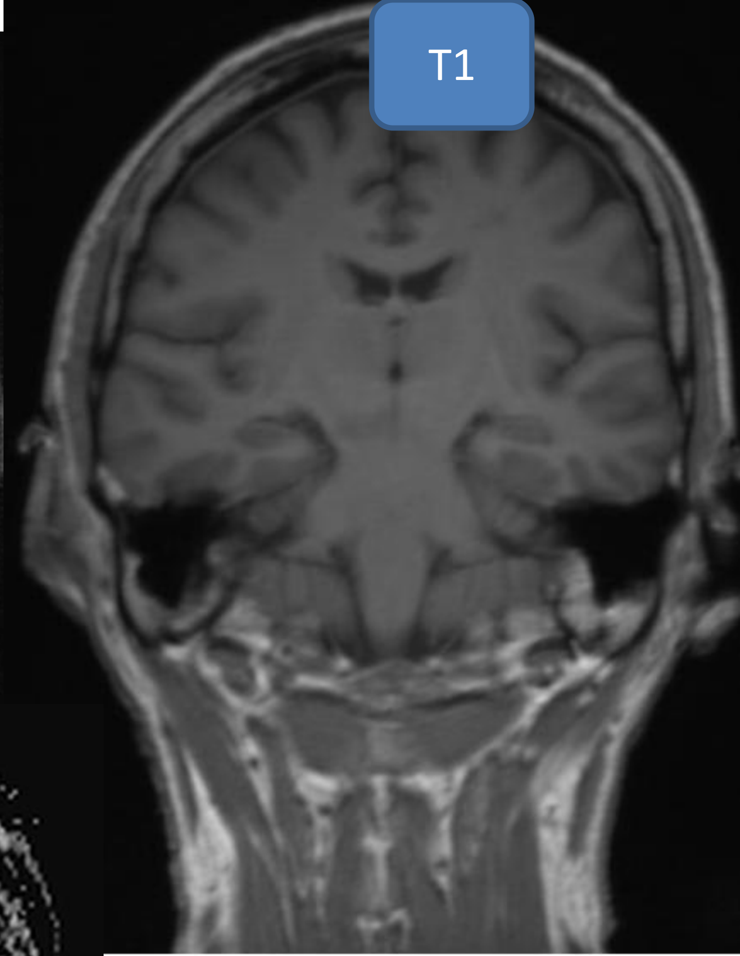


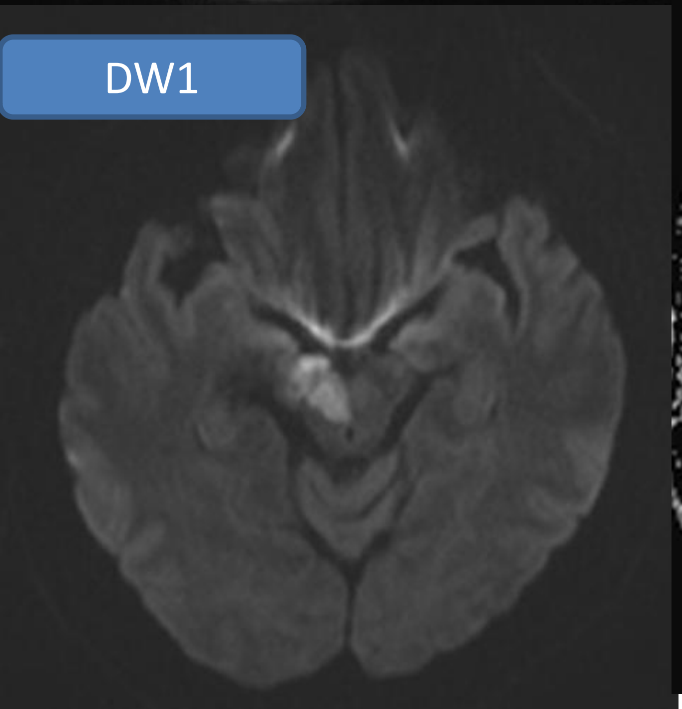


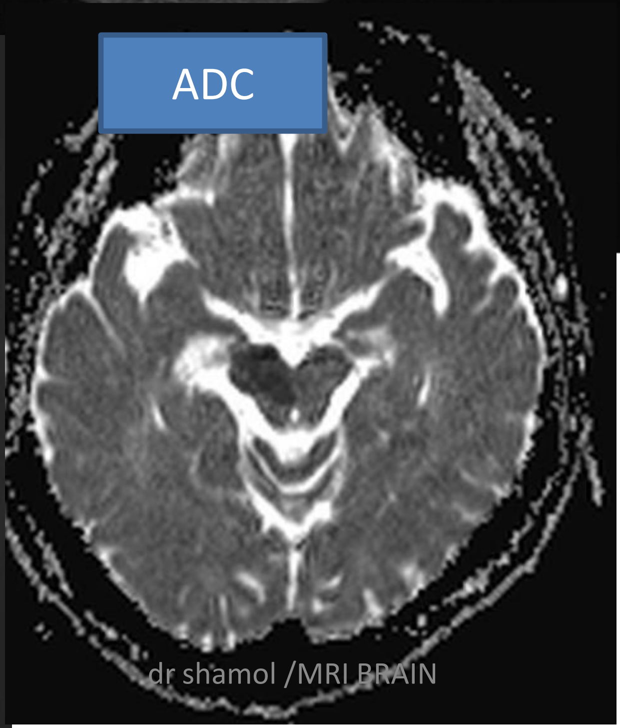
Well defined area of low T1 signal / high T2 signal with diffusion restriction in the left MCA territory. Loss of signal in the left MCA and its branches on the MRA.



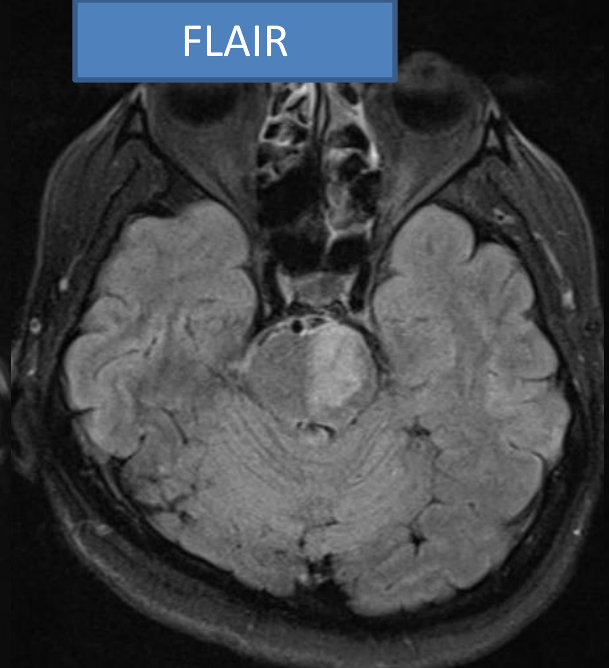


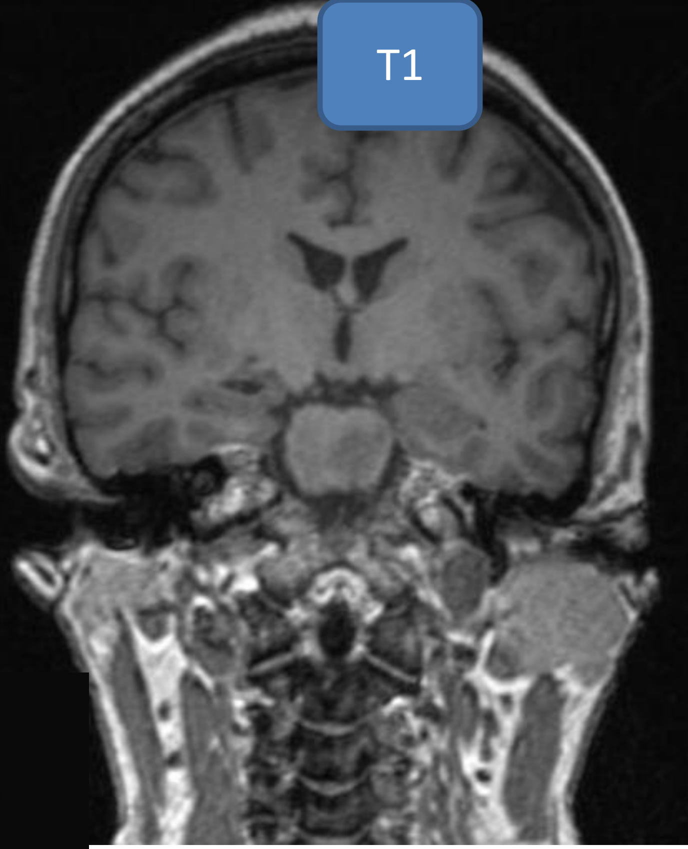


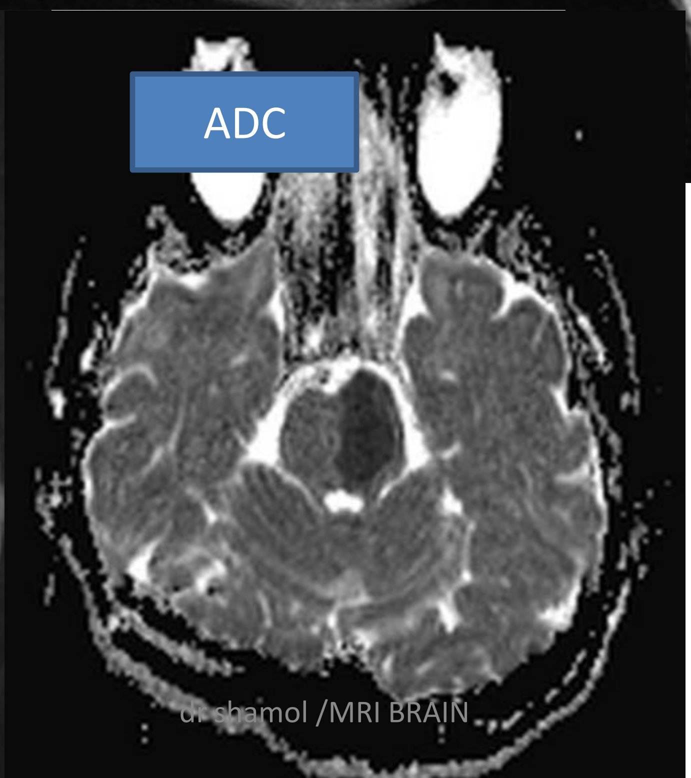




DWI/ADC map avid diffusion restriction in the right side of the mesencephalon, chiefly involving the tegmentum







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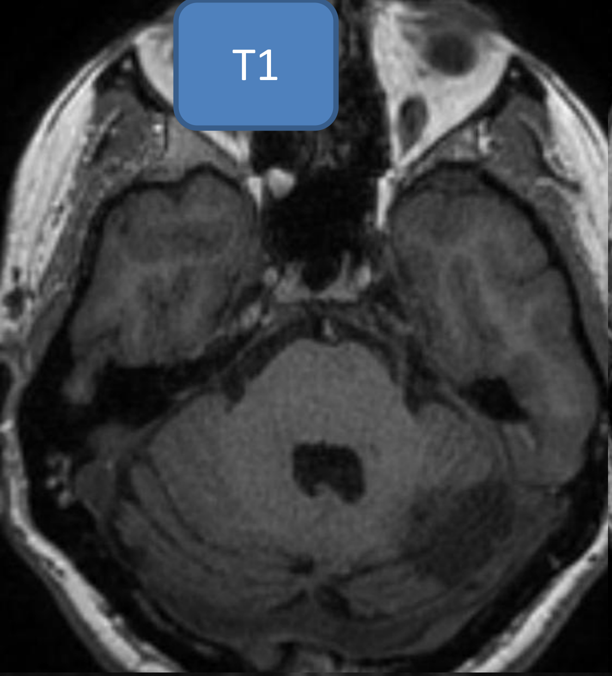
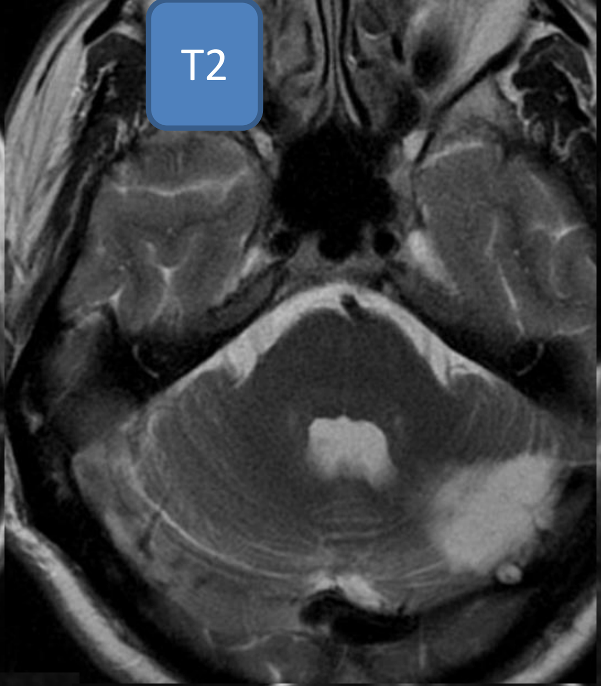
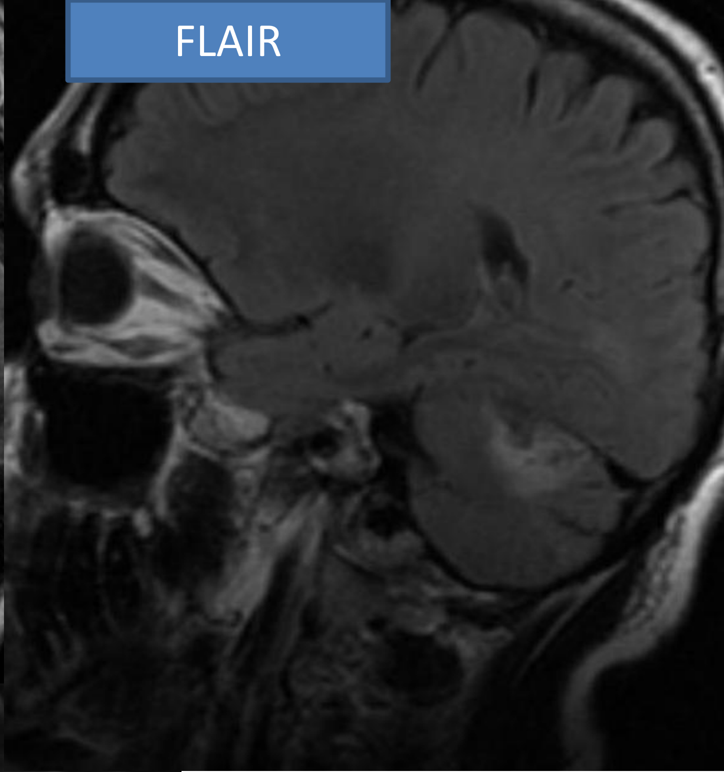
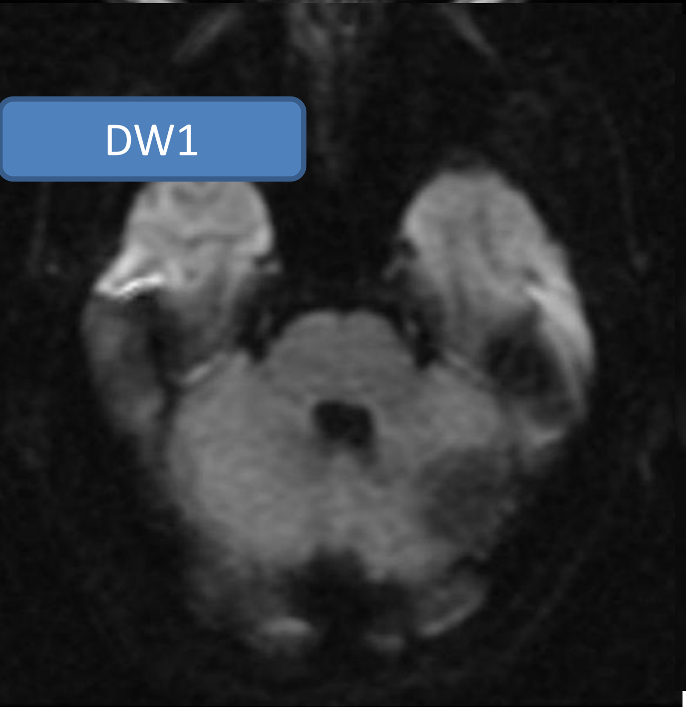
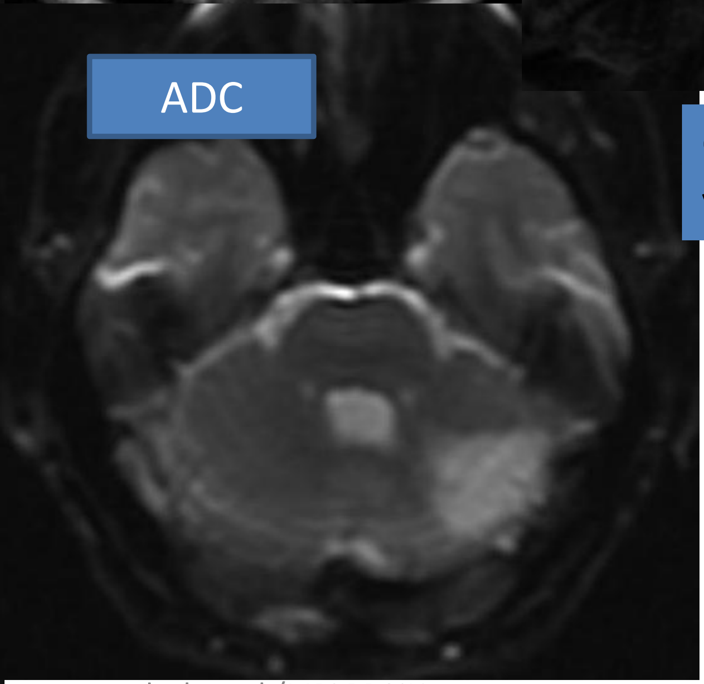


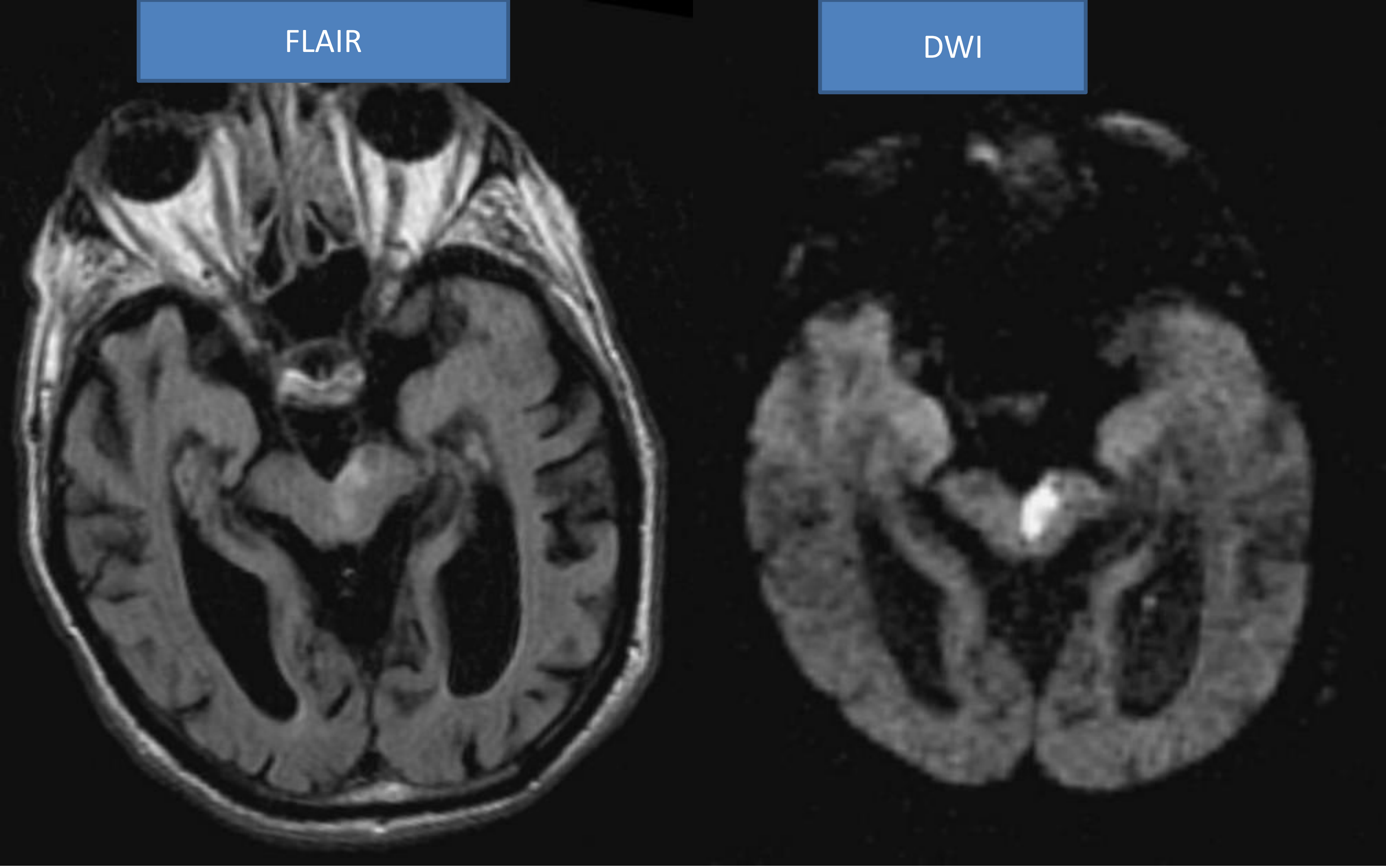


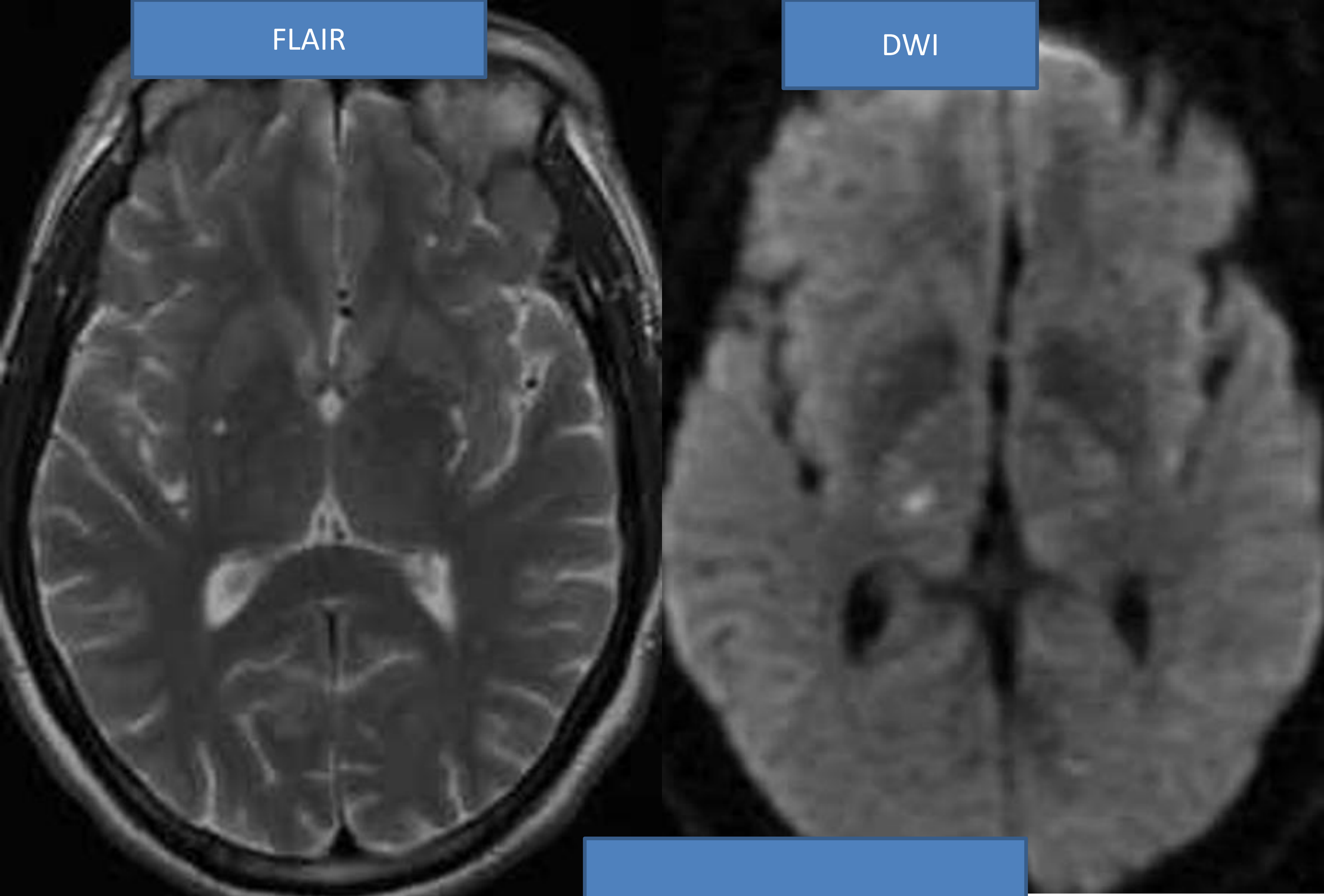






Old cerebellar stroke a year ago


FLAIR
DWI



Lacunar infarction

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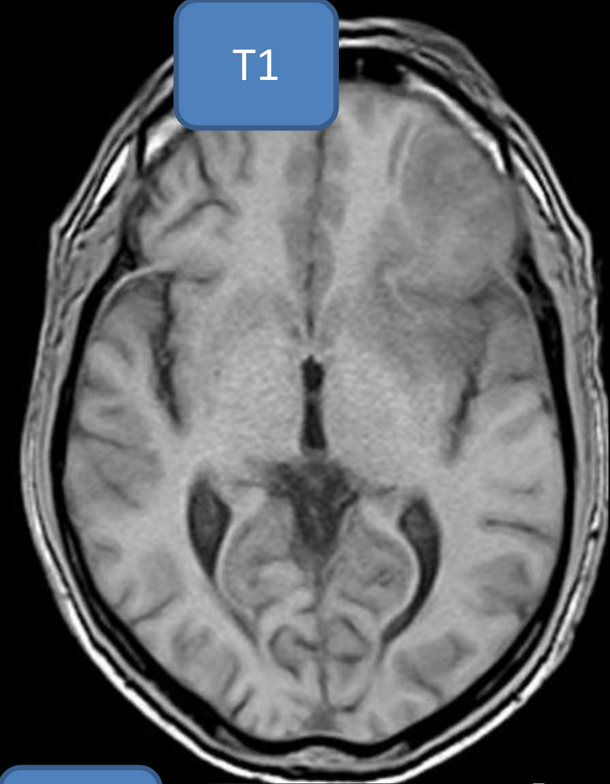
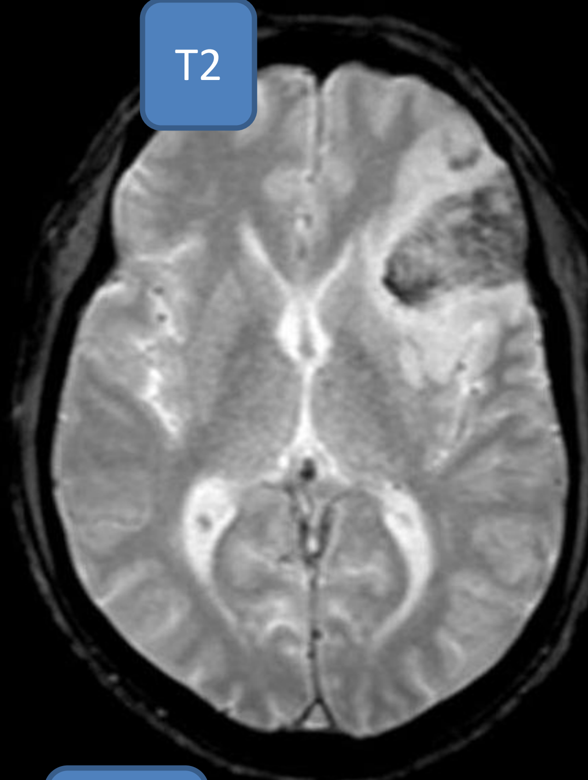
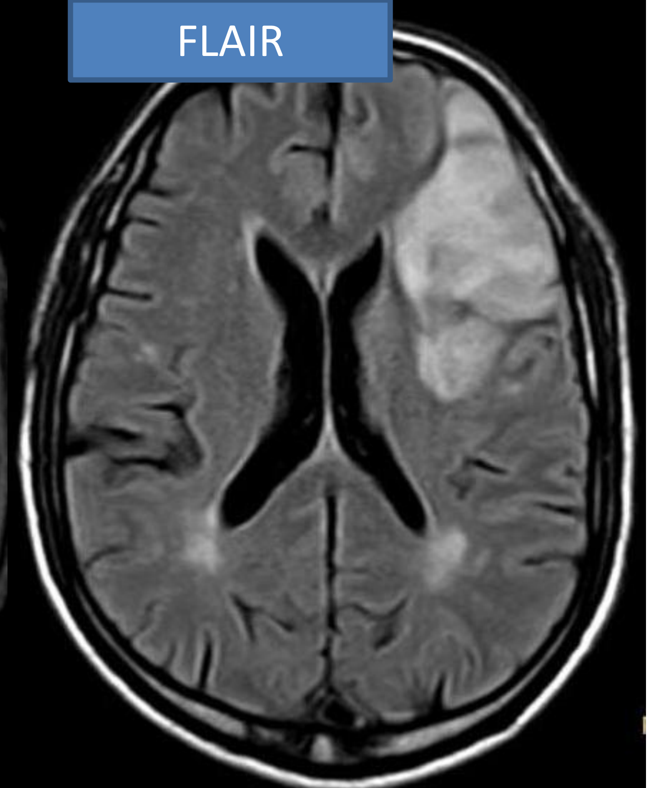
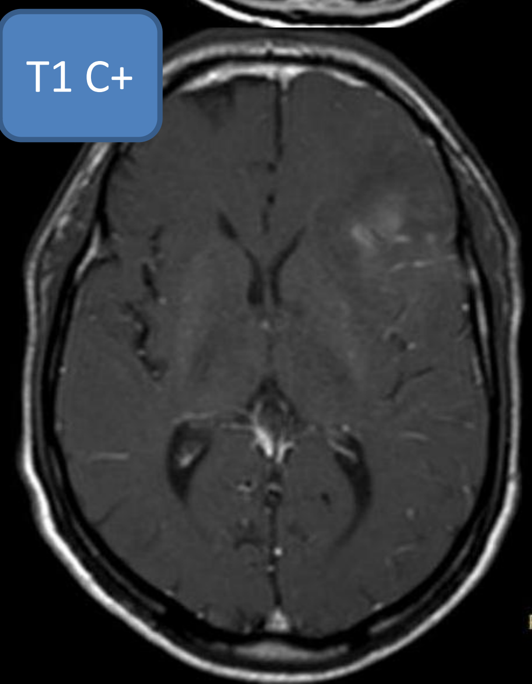
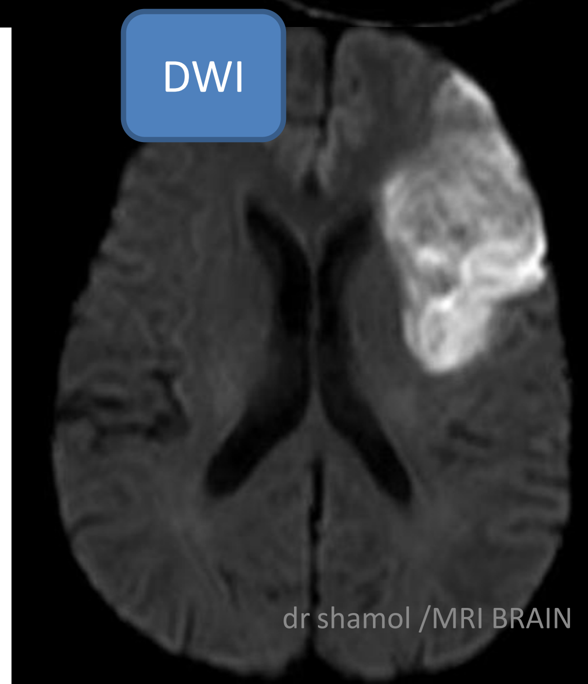








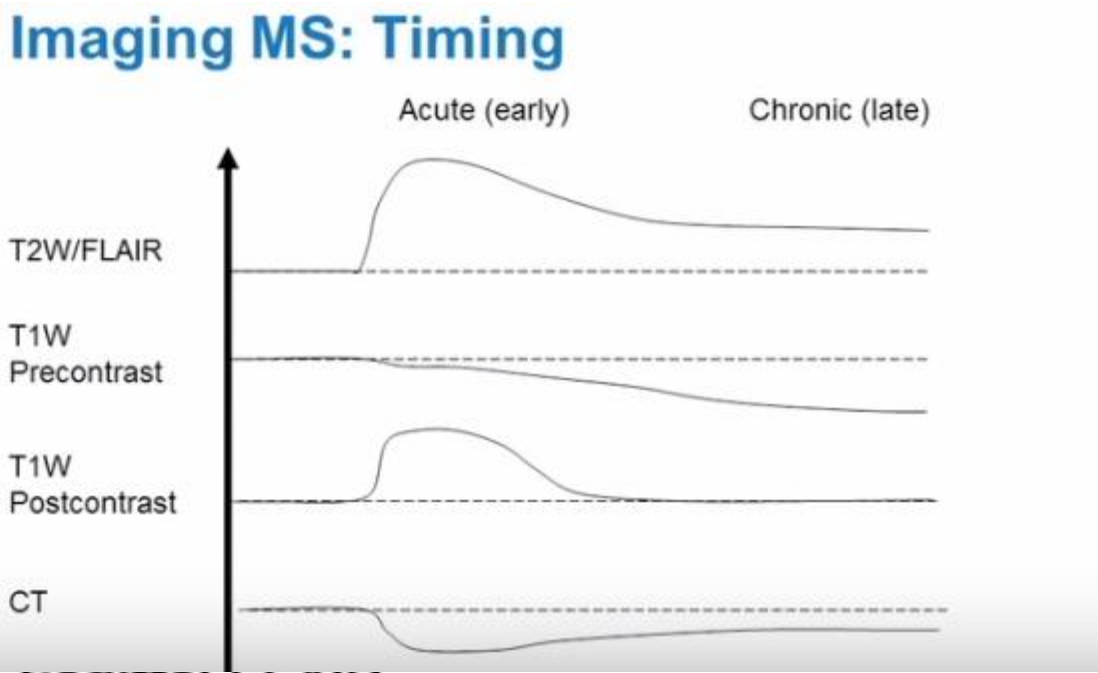


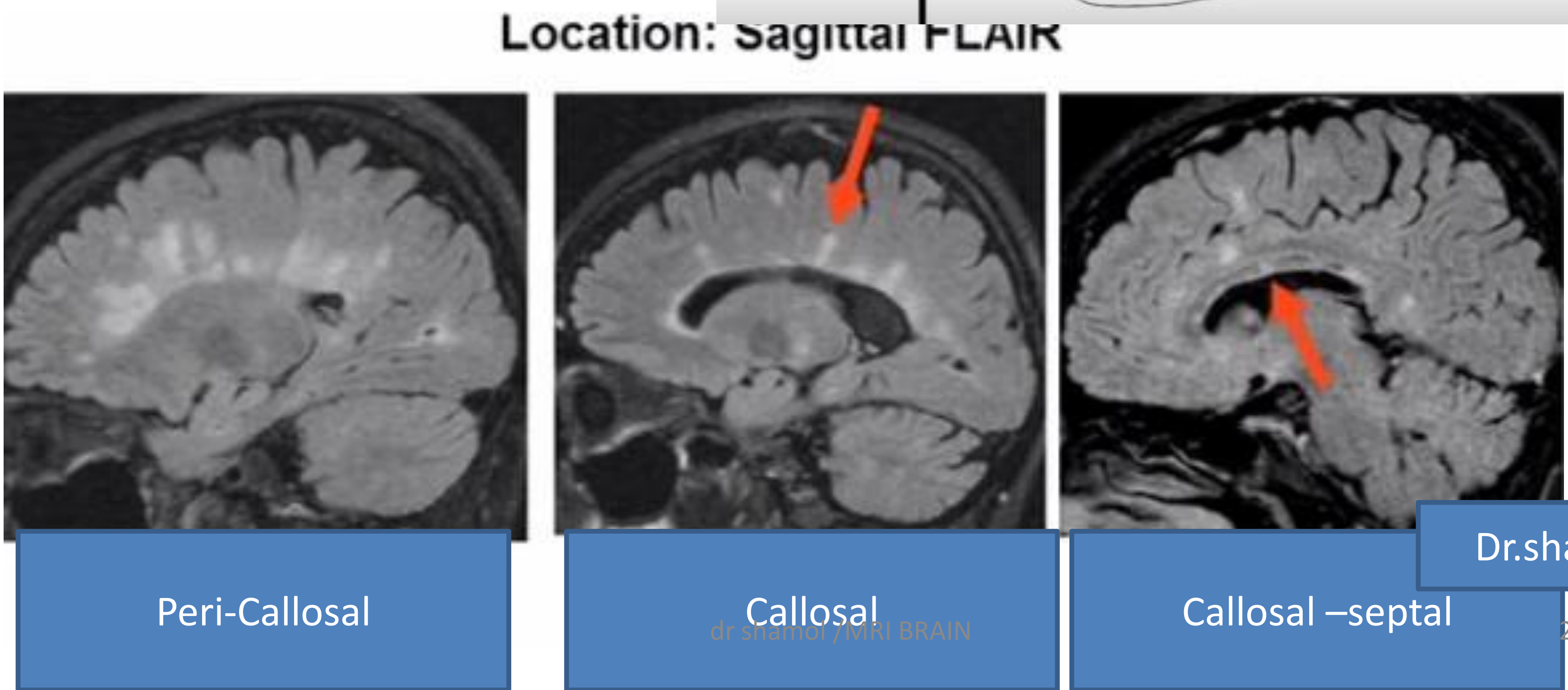
Left frontal subacute infarction that exhibits restricted diffusion in DWI, hypointense signal in T1 and hyperintense signal in FLAIR.
T2* images show hypointense area of hemosiderine deposition within the lesion

Haemorrhagic cerebral infarction

MS

Site of lesioin :
- ❖Periventricular
- ❖Callosal –septal margin
- ❖Corpus callosum
- ❖Pons
- ❖Anterior temporal lobe
- ❖Pons
- ❖Deep white matter
- ❖Cerebellum
- ❖Spinal cord





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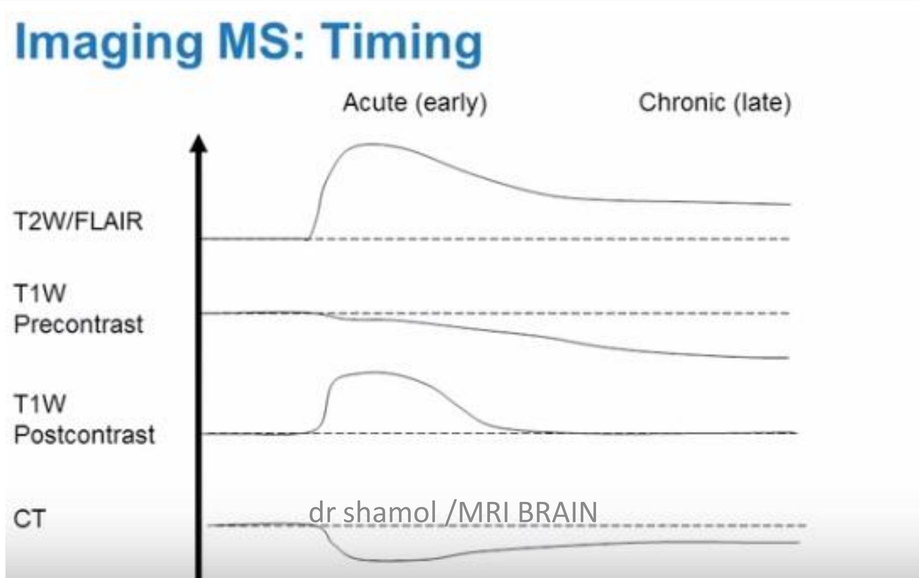
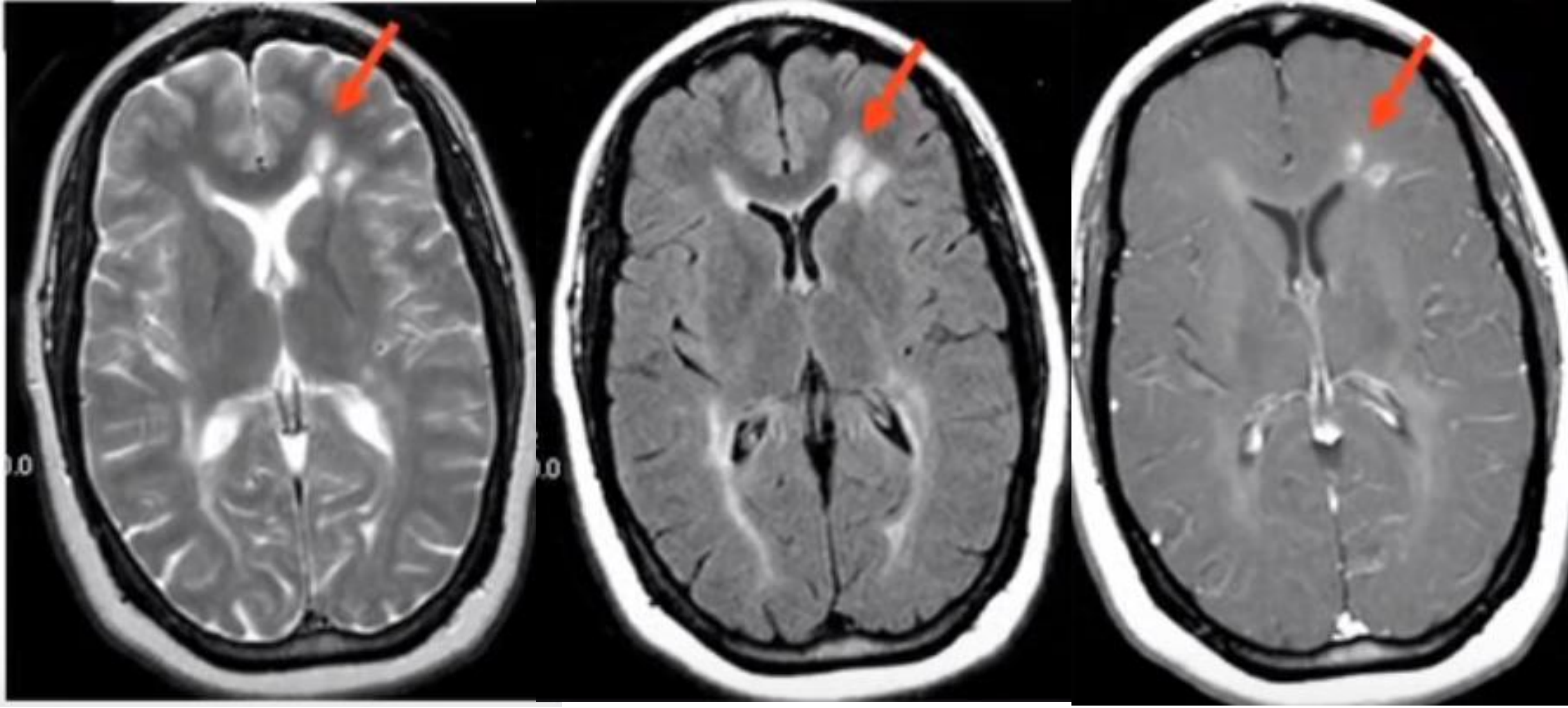
Phase	lesions are
T1	➢typically iso- to hypointense (T1 black holes)
T2	typically hyperintense
FLAIR	typically hyperintense
	more sensitive than T2 in detection of juxtacortical and periventricular plaques
	a very early sign is called "ependymal dot-dash sign": alternating small foci of hyperintensity along the callososeptal interface
	when these propagate centrifugally along the medullary venules and arranged perpendicular to the lateral ventricles in a triangular configuration (extending radially outward - best seen on parasagittal images), they are termed Dawson's fingers
T1 C+ (Gd)	active lesions show enhancement
	enhancement is often incomplete around the periphery (open ring sign)



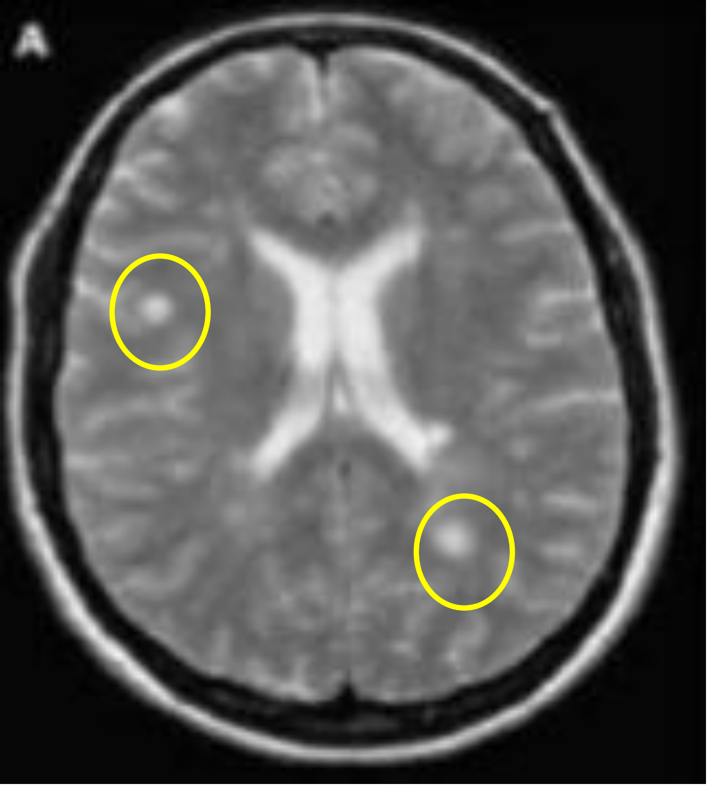


T2

FLAIR

T1 C

HYPER INTENSE LESION OF MS



T2-weighted (A).

post-contrast T1-weighted (B)

A shows Two hyperintense lesion in T 2 .
Two of them enhanced in B with gadolinium-DTPA.
This is a sign of increased blood–bran barrier permeability and continuing inflammation.

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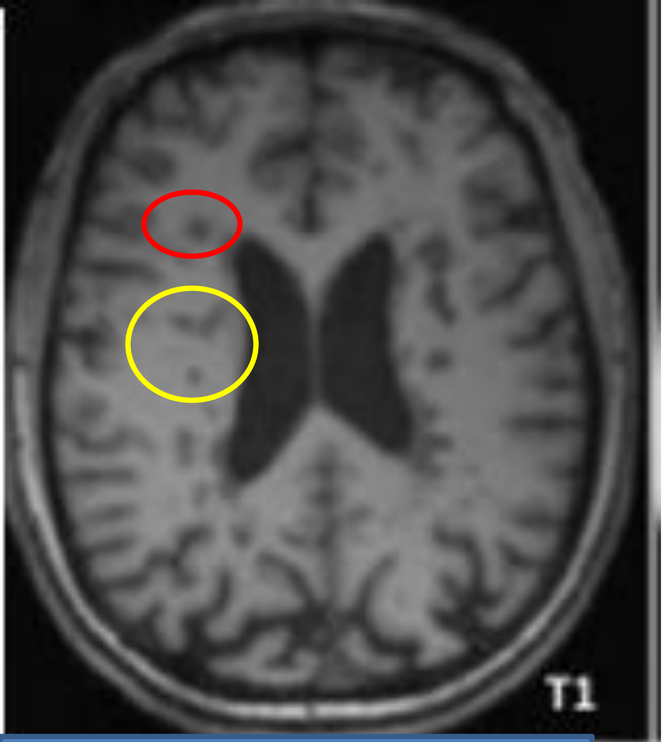
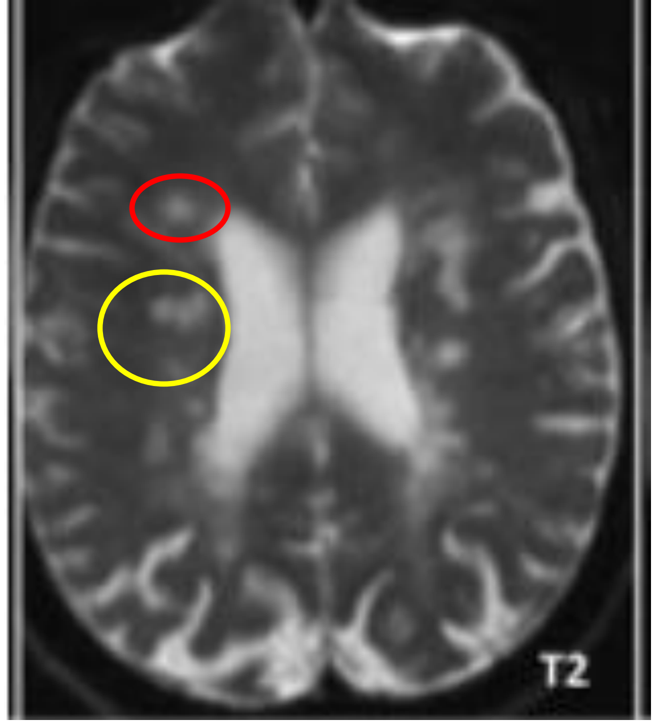


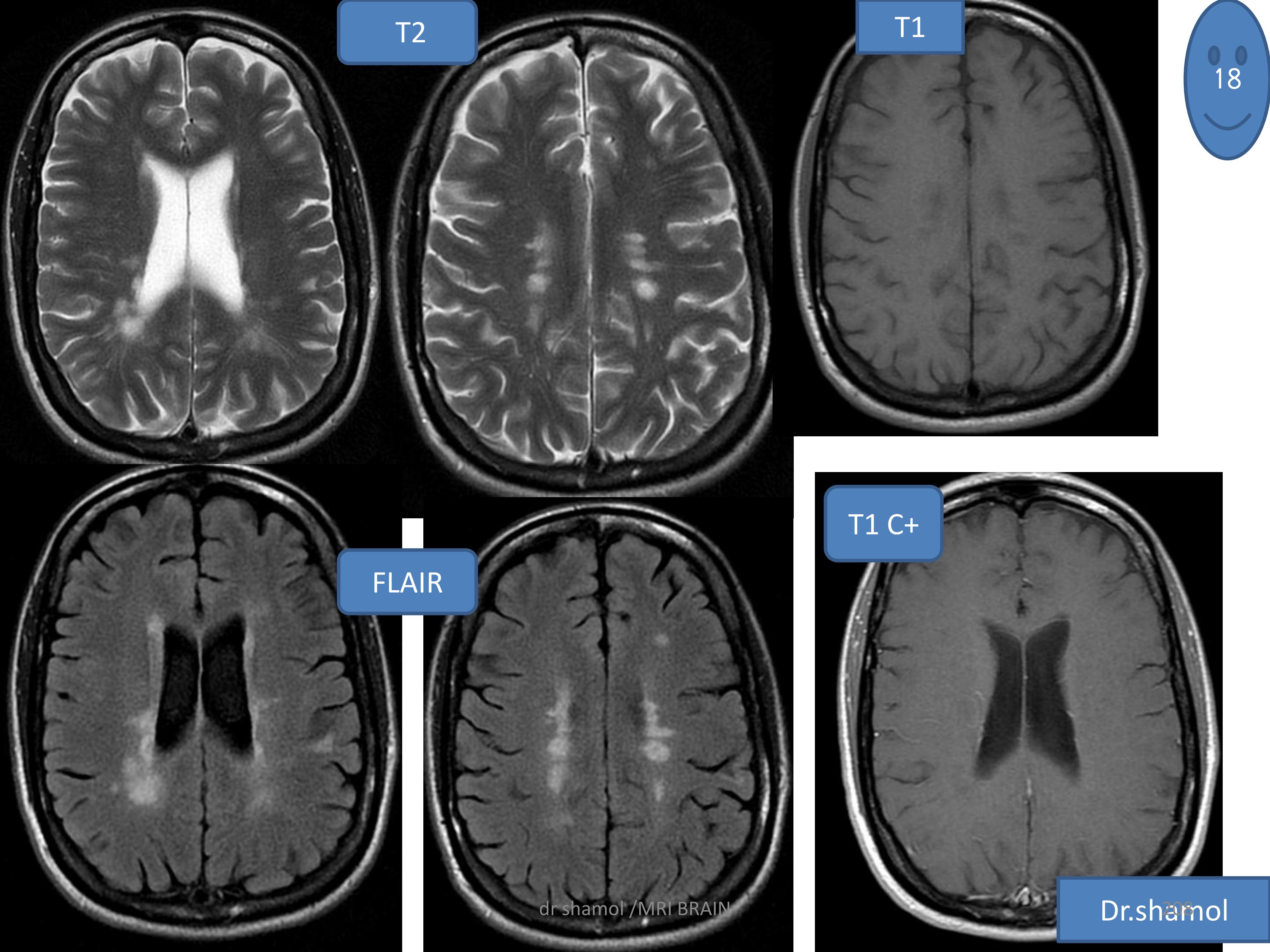
Hypointense periventricular lesion



Hyperintense Periventricular lesion

Post contrast enhancing lesion

Case MS -1



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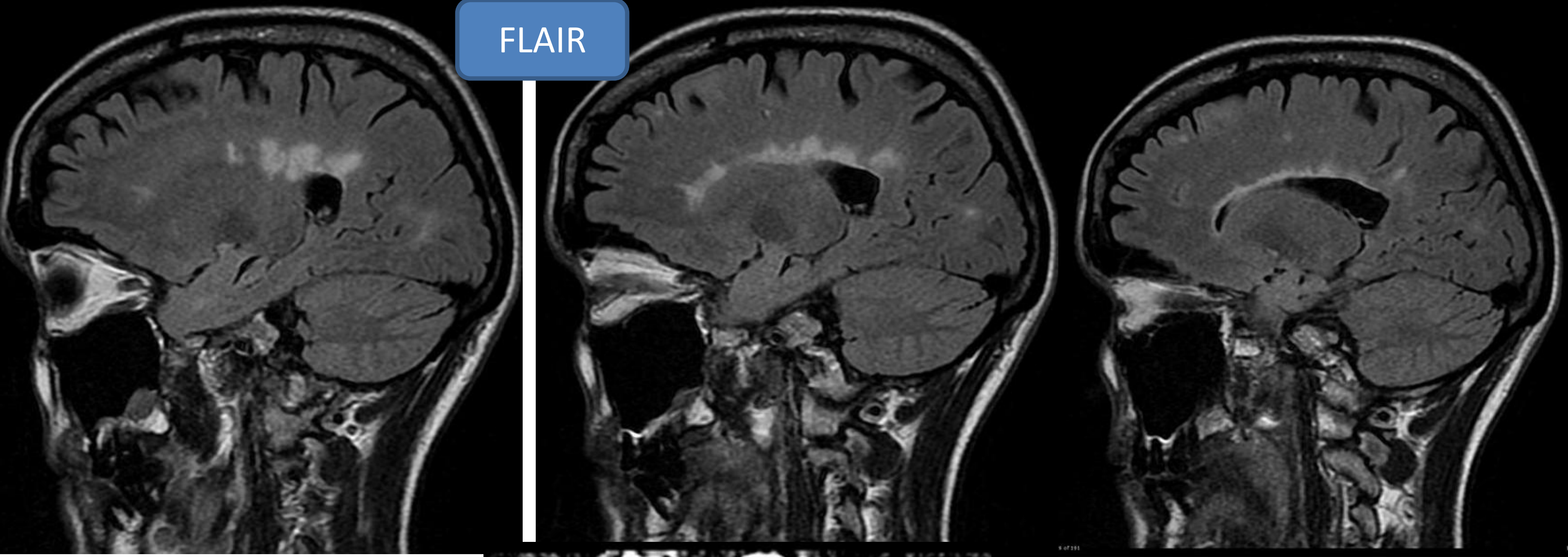

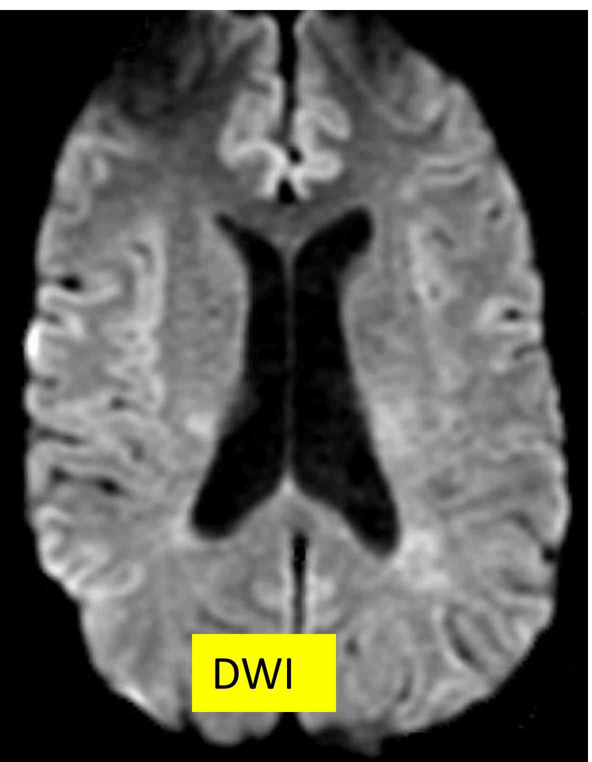

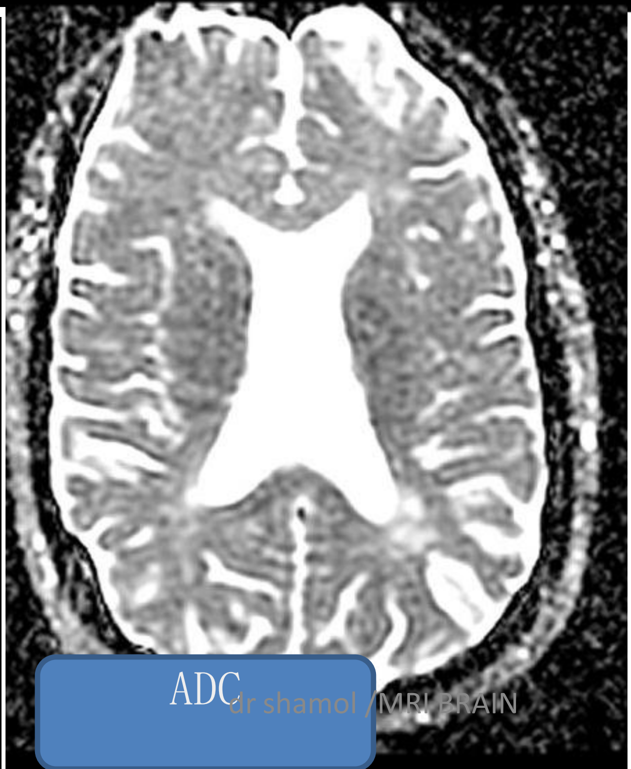

Widespread white matter T2 regions of hyperintensity are seen bilaterally, particularly in a periventricular distribution, with appearance is characteristic of multiple sclerosis. No enhancement or restricted diffusion to suggest current/recent activity.

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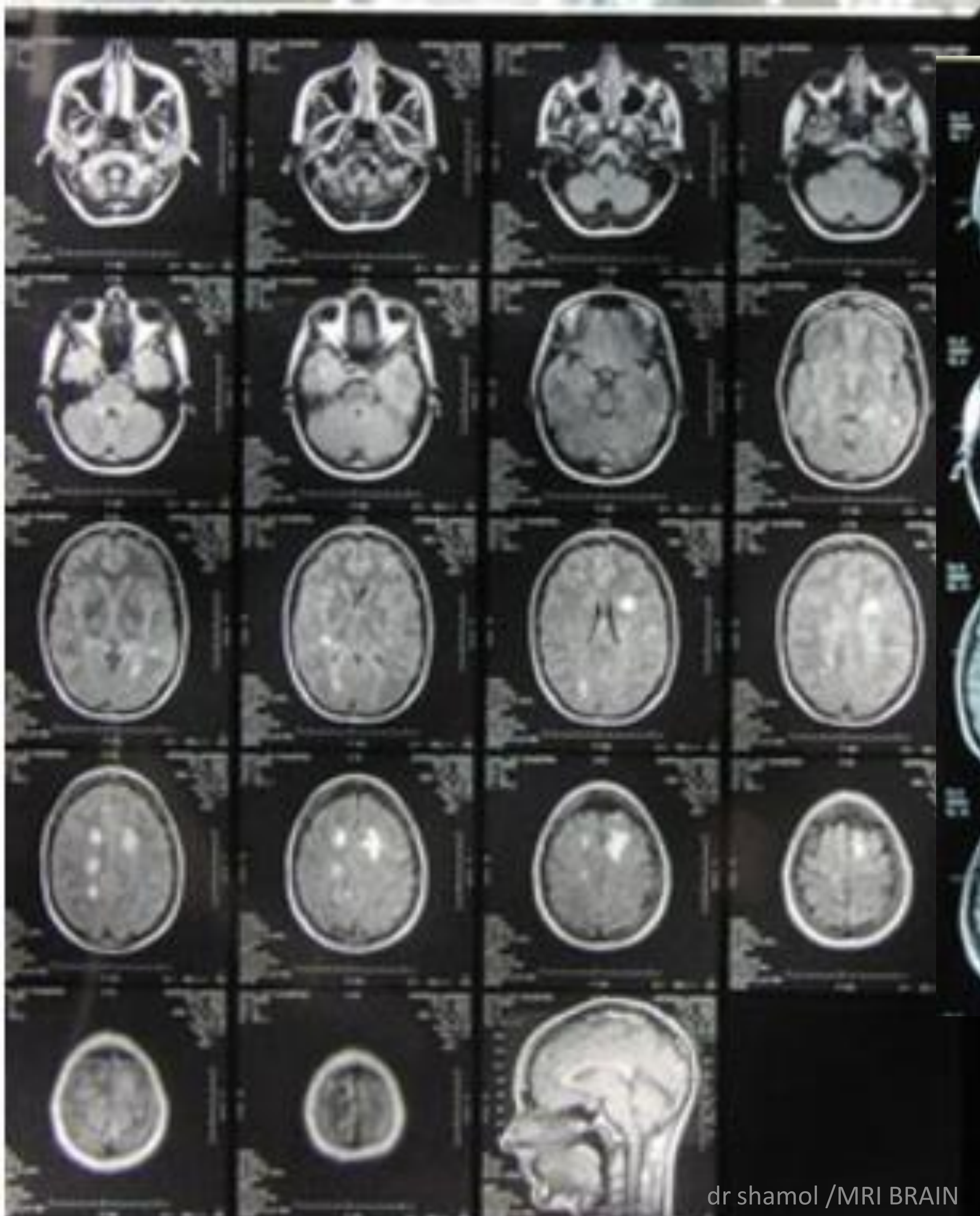
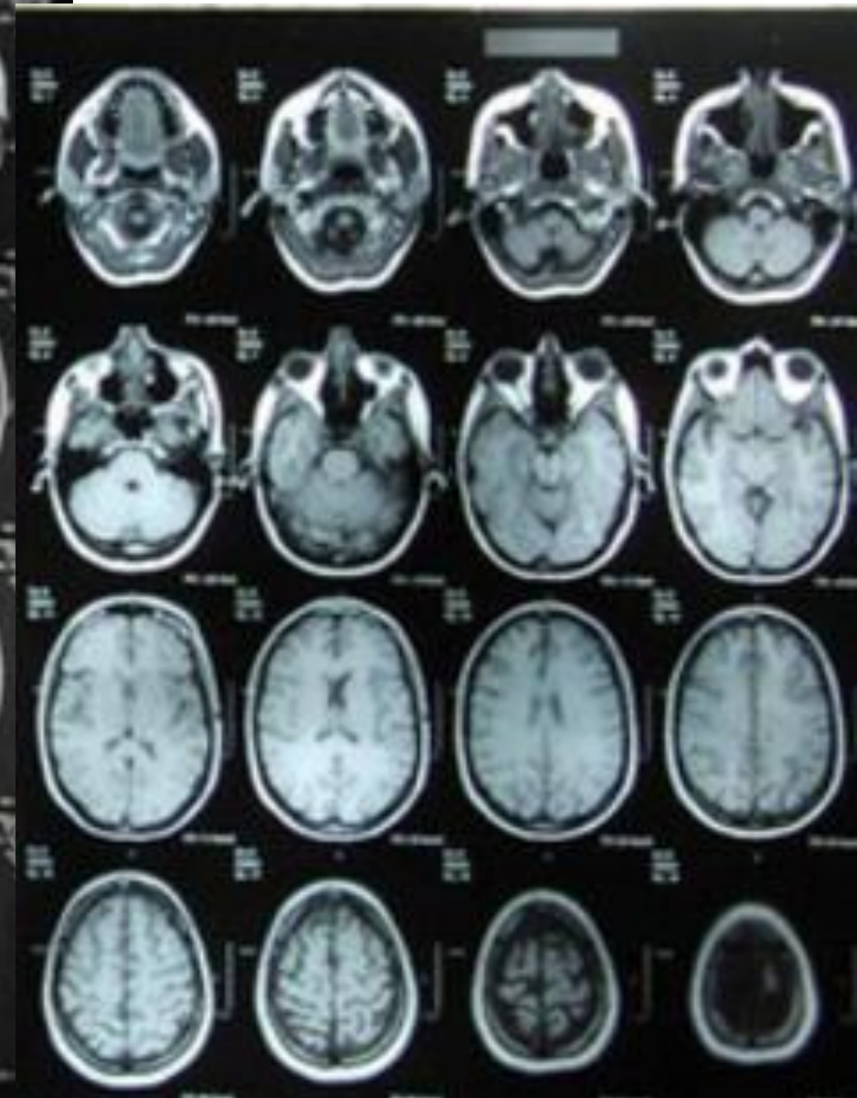
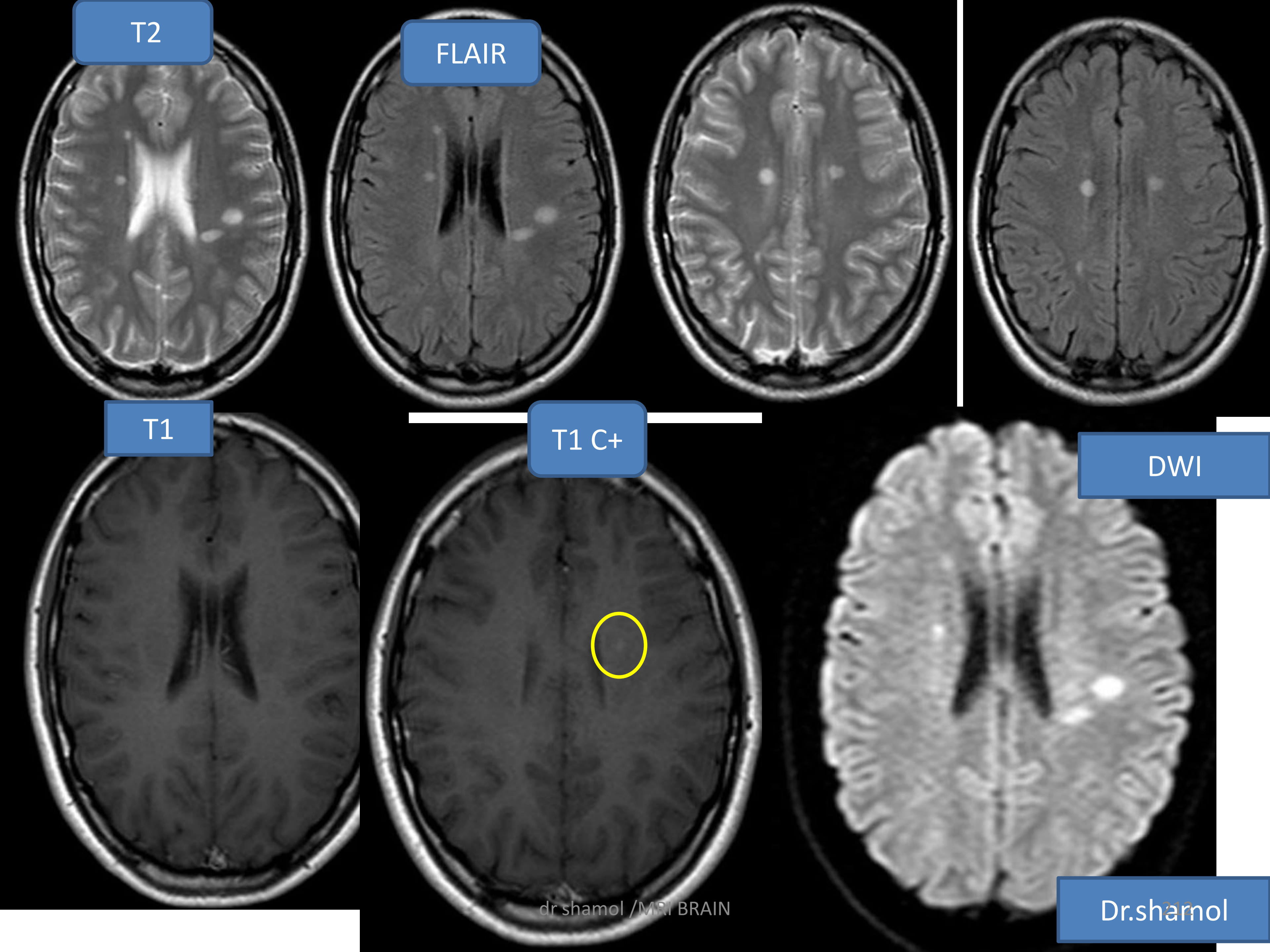
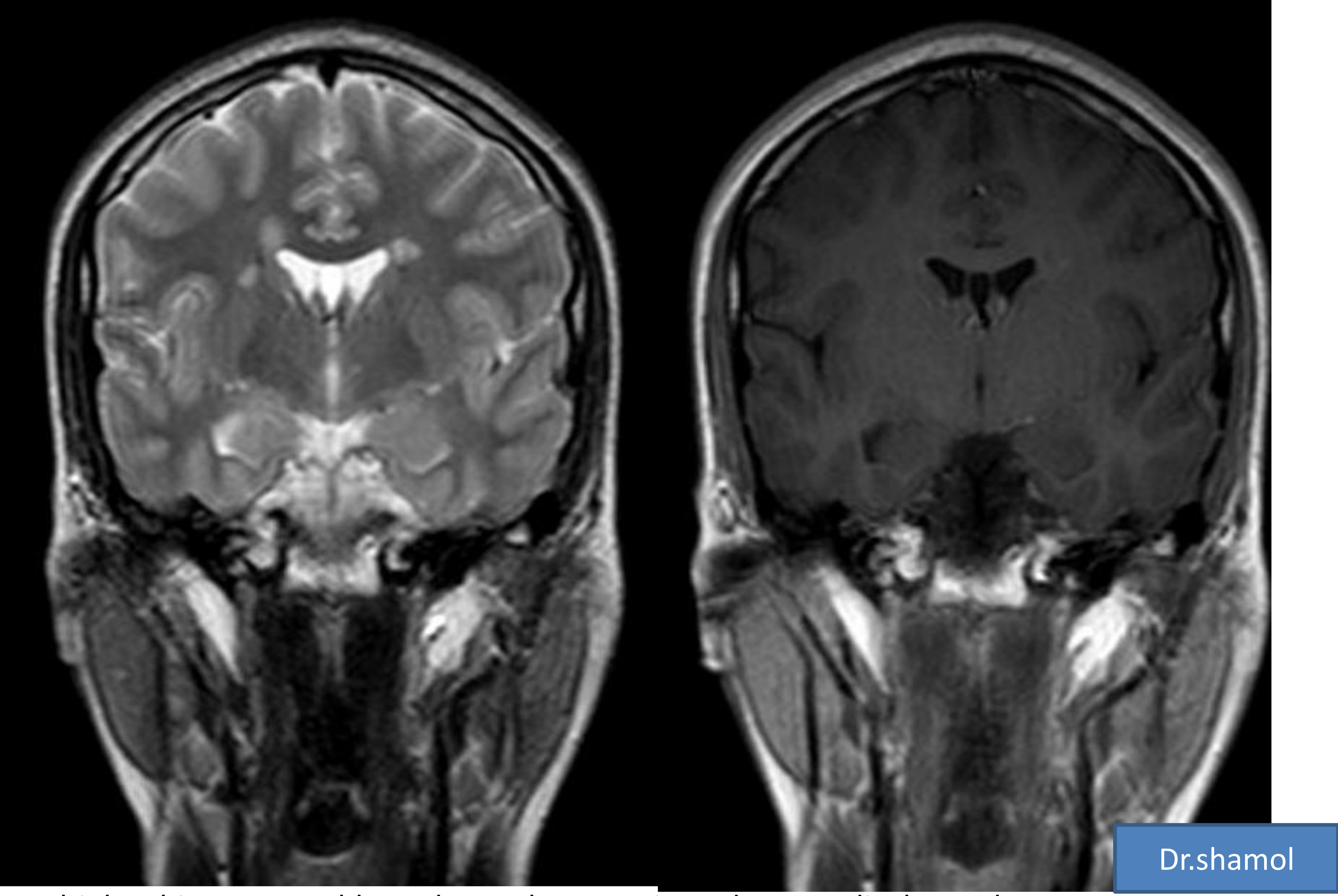




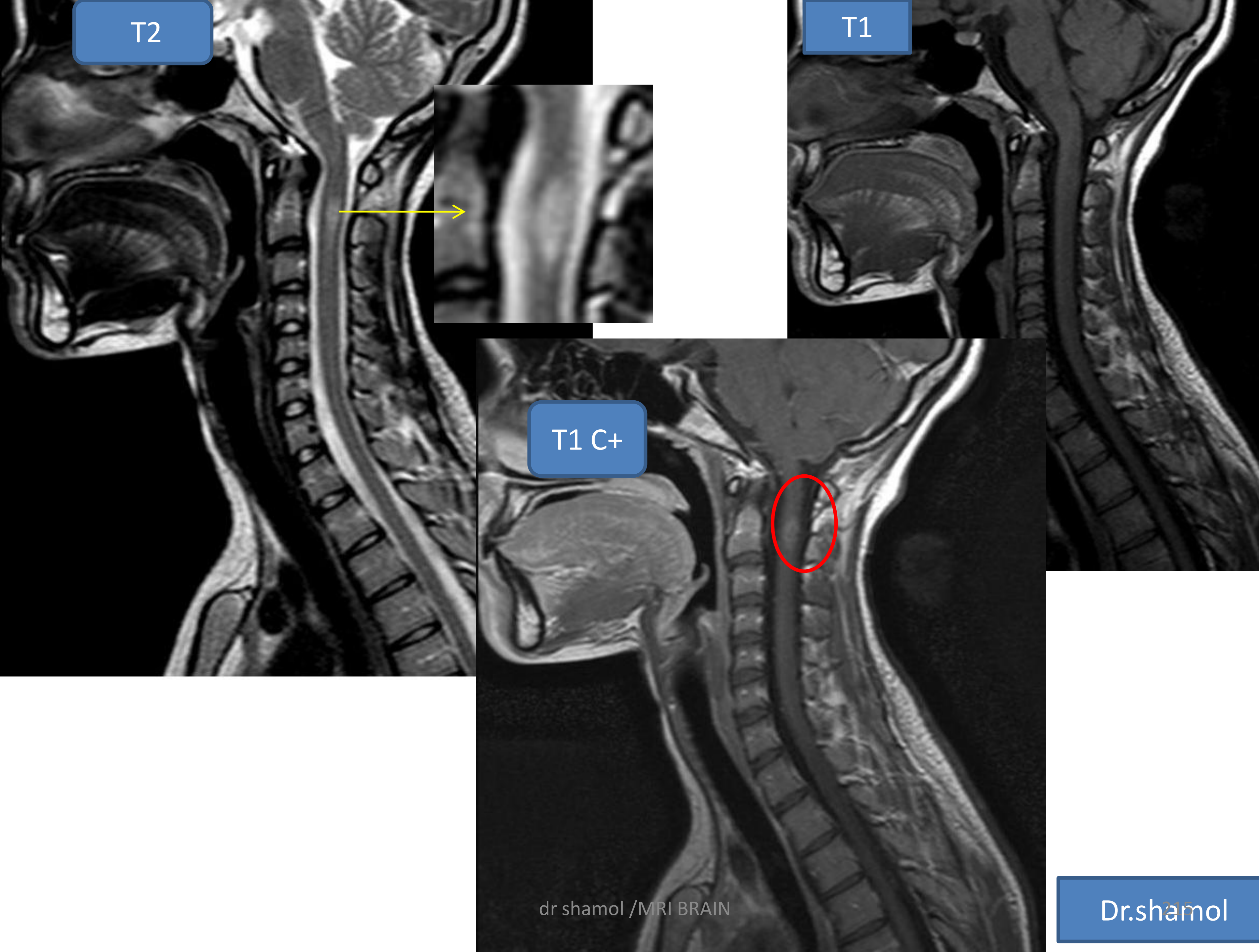
Case MS -2

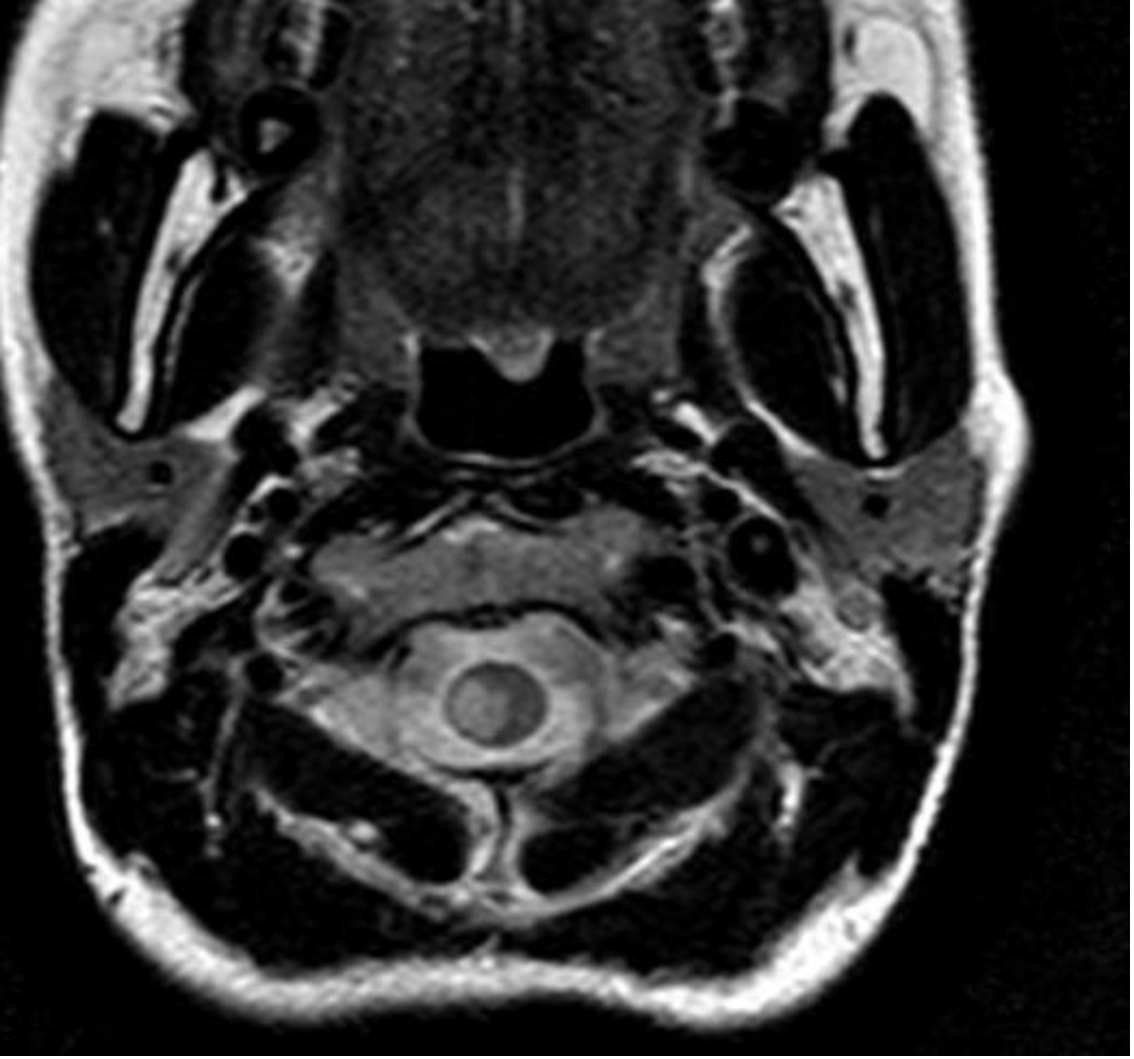

T2
FLAIR
T1
T1 C+
DWI
Dr.shamol

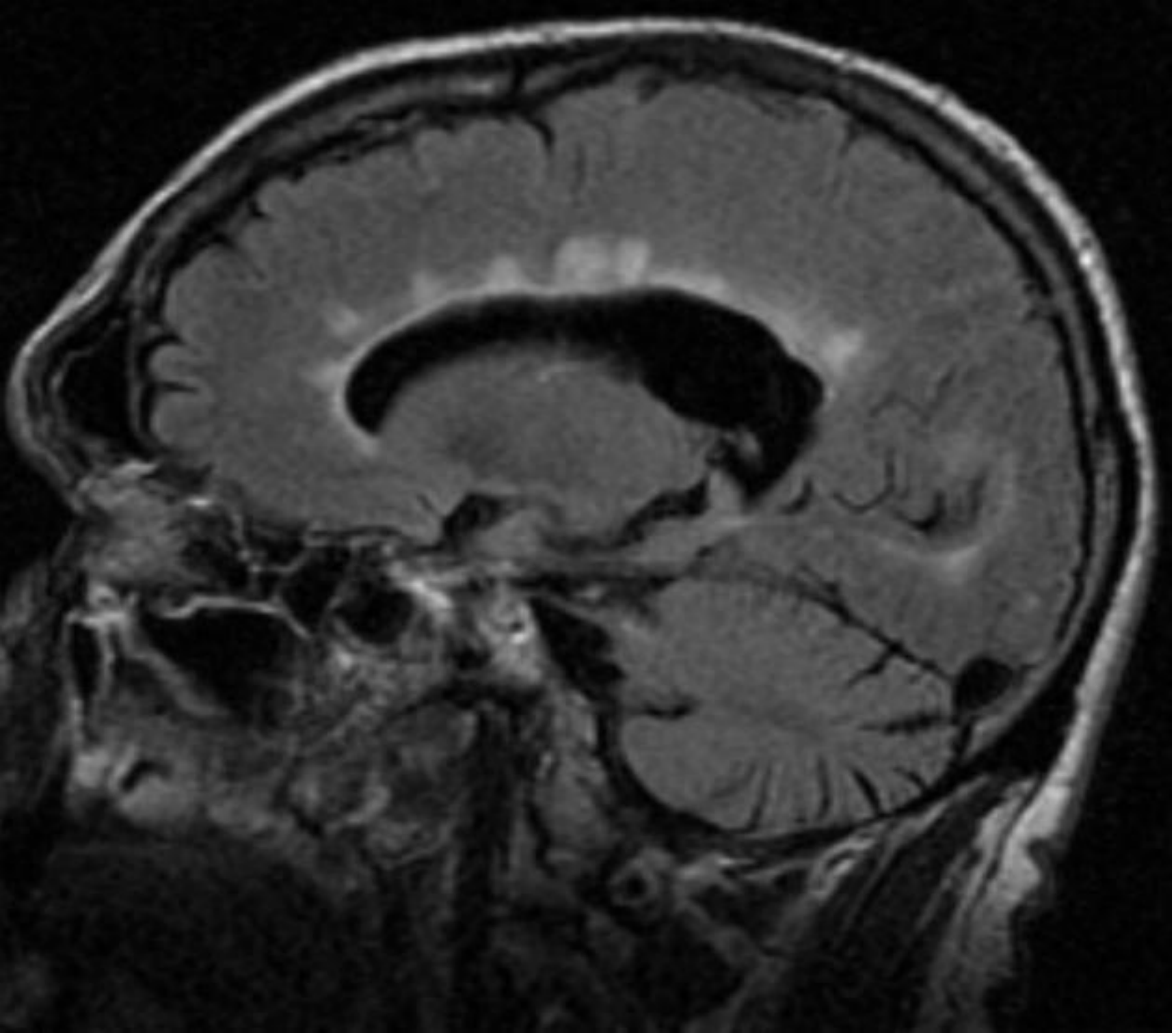


Multiple white matter oblong shape plaques oriented perpendicular to the corpus callosum (Dawson fingers) and display bright signal in T2, FLAIR and DWI. One of them shows faint enhancement in post contrast scan.

Case MS -3

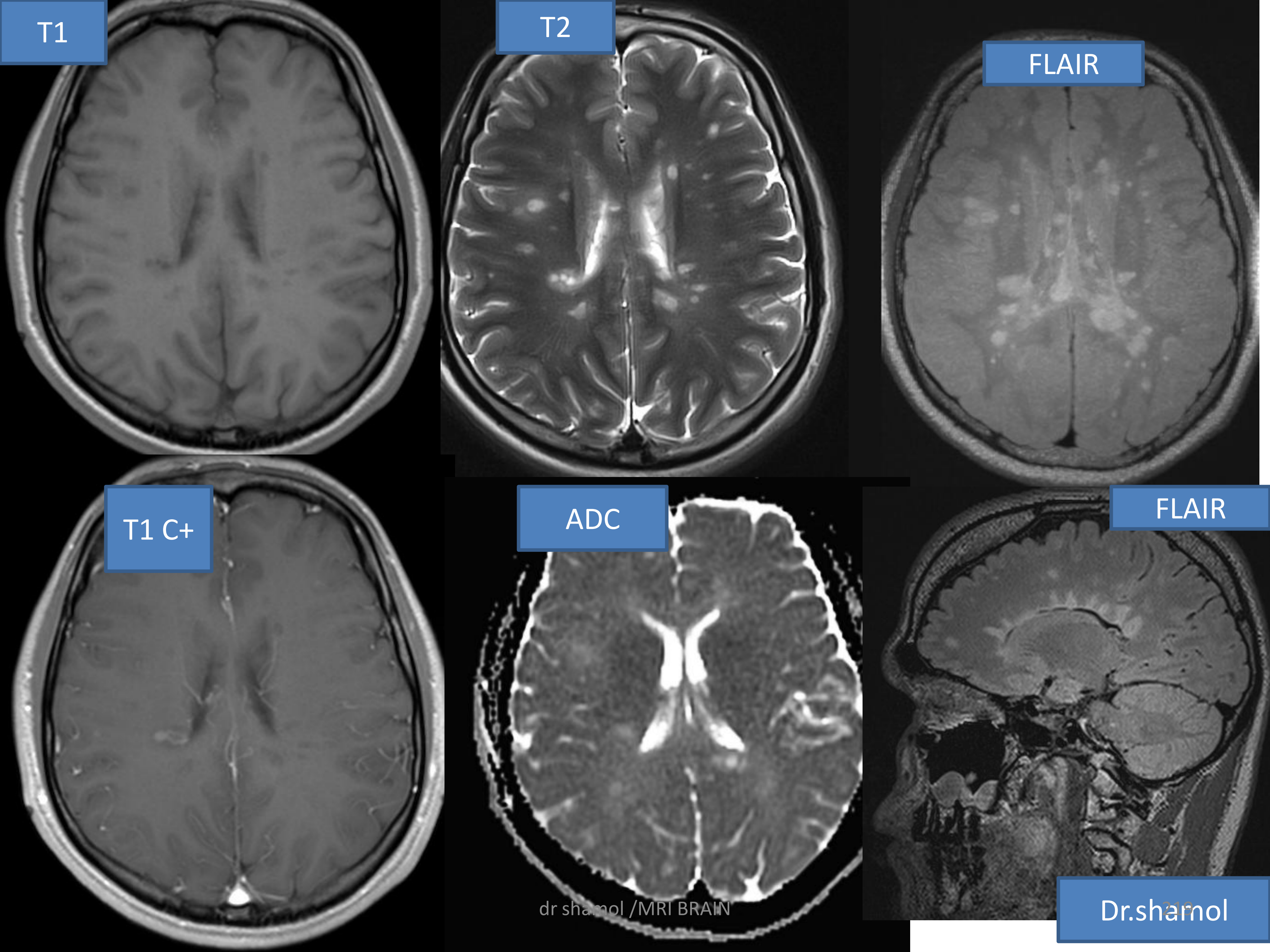

T2
T1
T1 C+

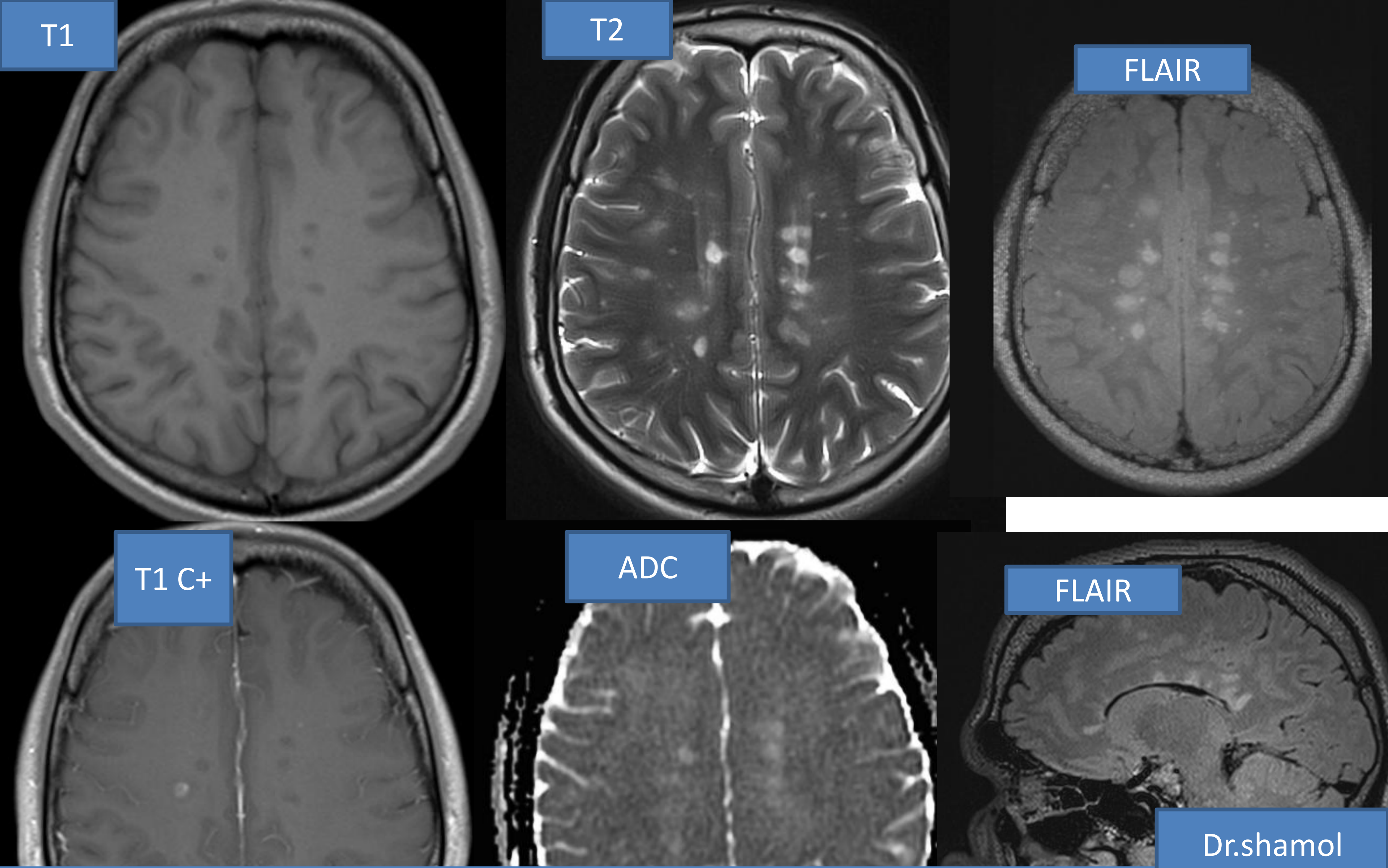




Dawson's fingers: MS plaques extending up through corpus callosum.

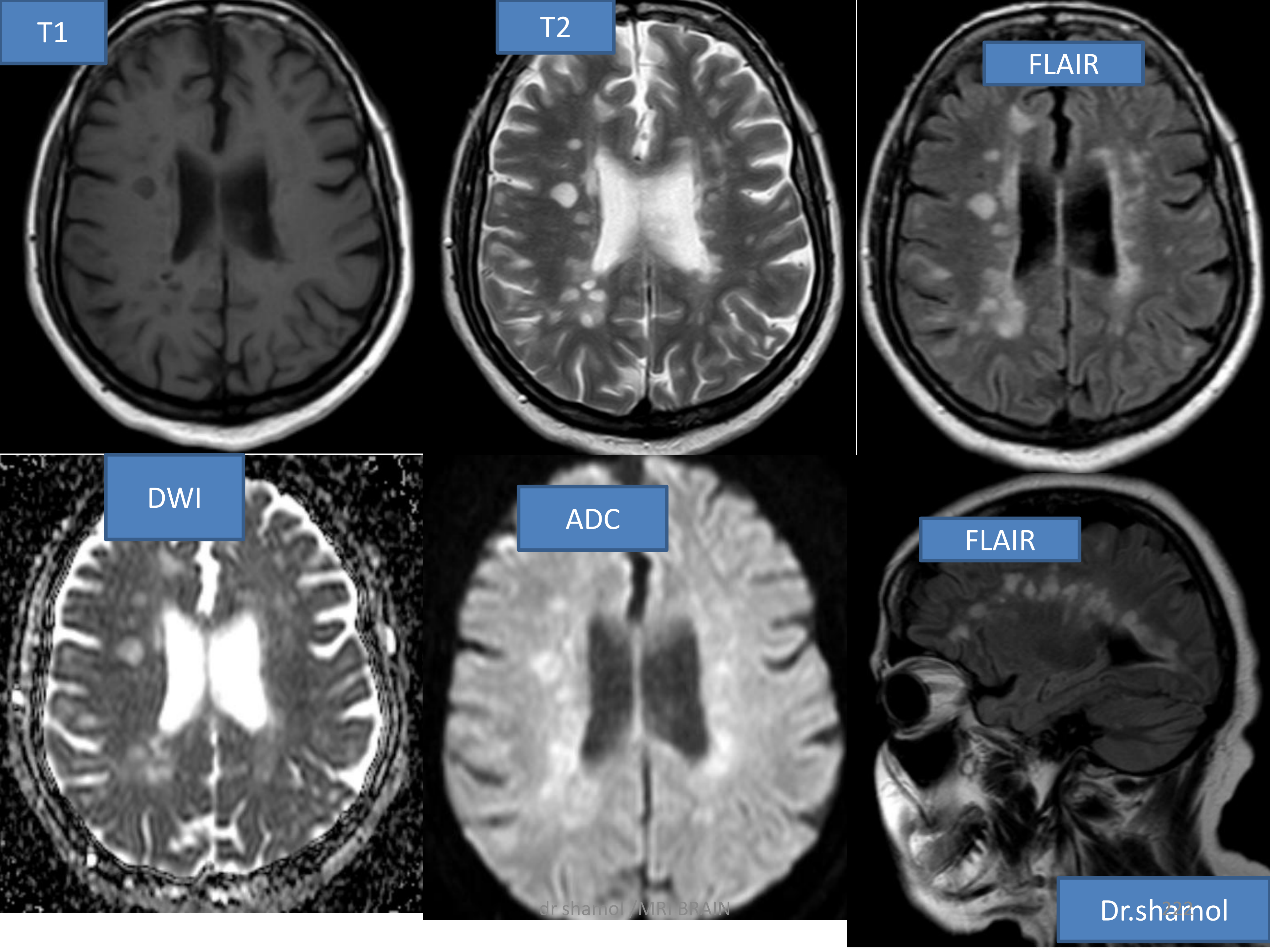
Case MS -4

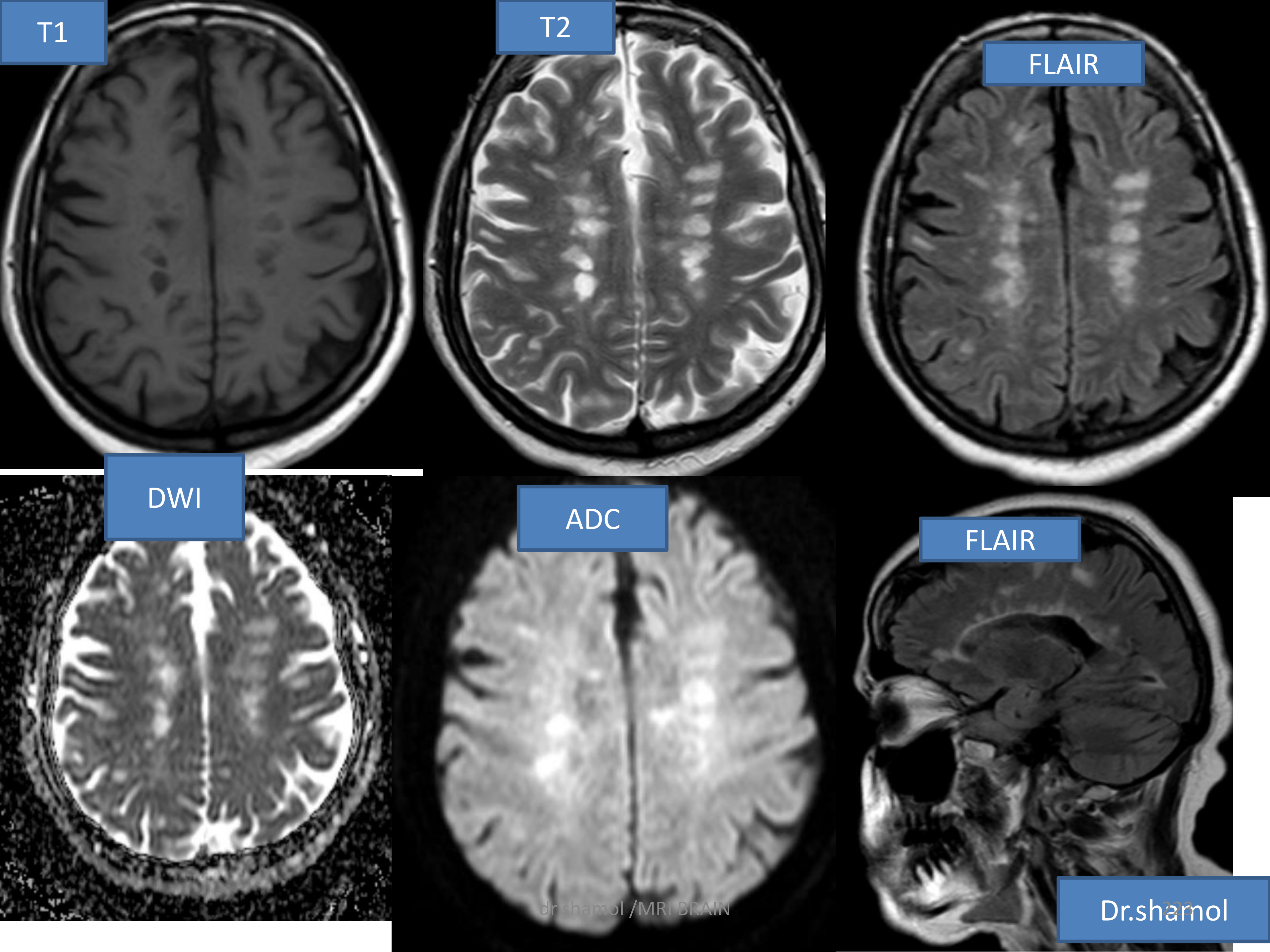

T1
T2
FLAIR
T1 C+
ADC
FLAIR
Dr.shamol

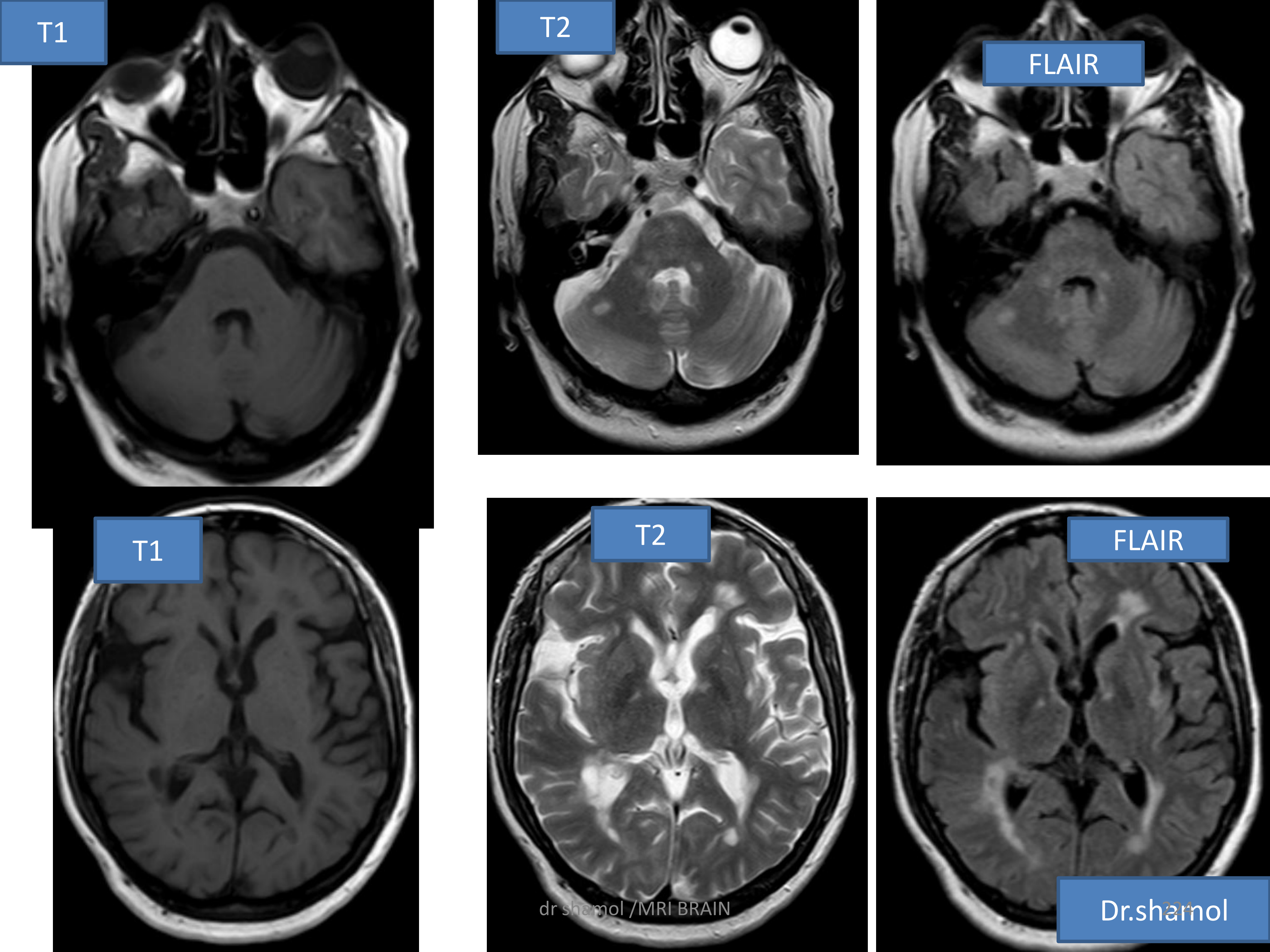


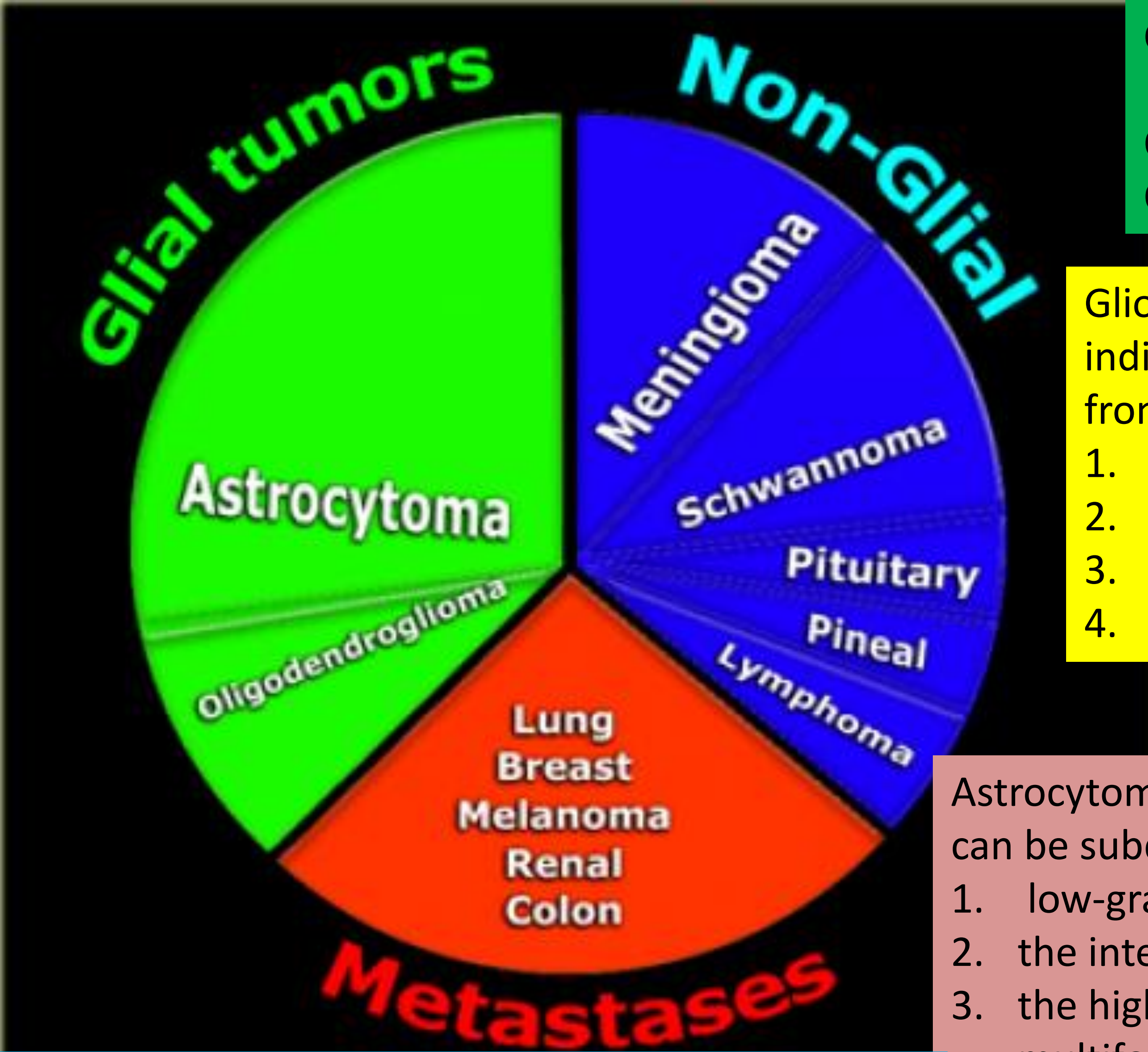
Multiple scattered supra and infratentorial white matter plaques, most of them are seen at the periventricular region perpendicular to the lateral ventricles giving the appearance of Dawson fingers, Most of the lesions show T2 shine through effect on DWI/ADC. Characteristically they display low signal on T1 WIs.

BLACK HOLE


T1
T2
FLAIR
DWI
ADC
FLAIR
Dr.shamol


T1
T2
FLAIR
DWI
ADC
FLAIR
Dr.shamol


T1
T2
FLAIR
T1
T2
FLAIR
Dr.shamol



CNS tumors
One-third are metastatic
One third are gliomas
One-third is of non-glial origin.

Glioma is a non-specific term indicating that the tumor originates from glial cells like

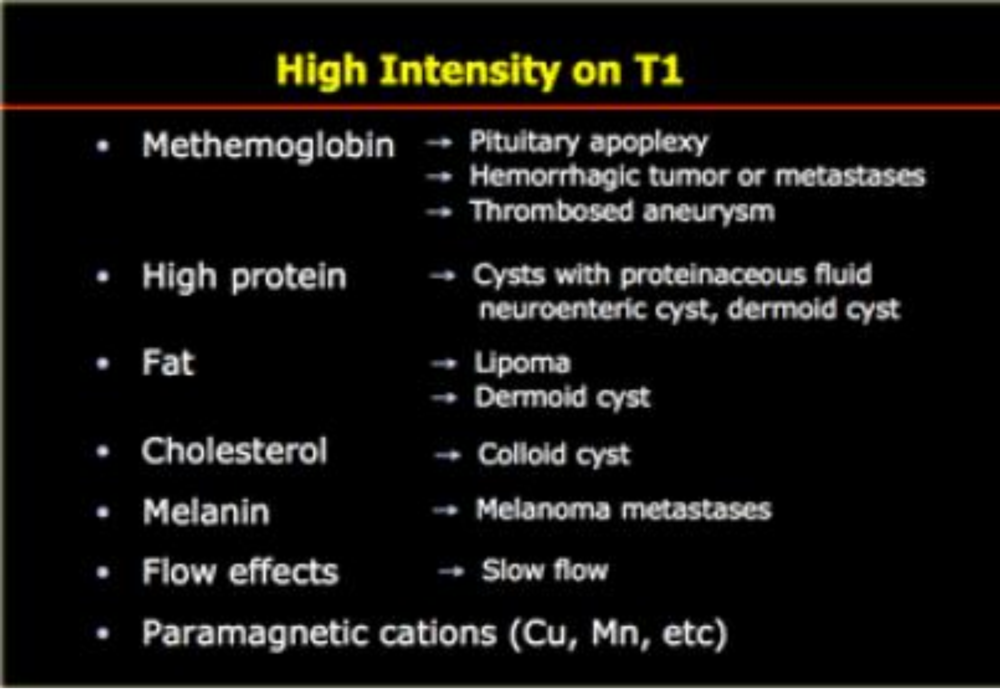
1. astrocytes,
2. oligodendrocytes,
3. ependymal and
4. choroid plexus cells.

Astrocytoma is the most common glioma and can be subdivided into the

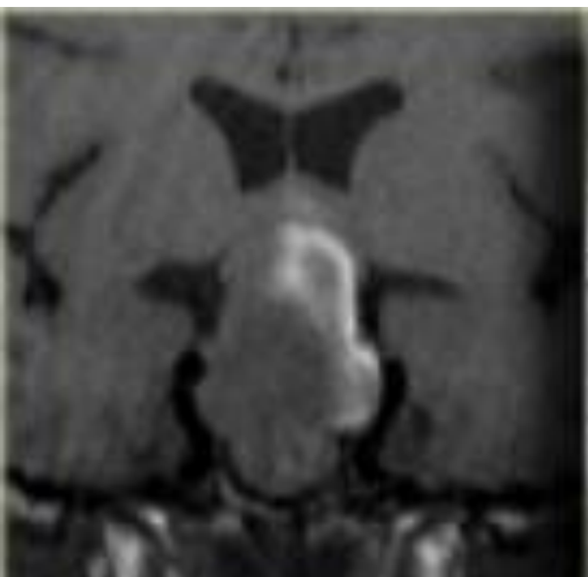
1. low-grade pilocytic type,
2. the intermediate anaplastic type and
3. the high grade malignant glioblastoma multiforme (GBM). is the most common type (50% of all astrocytomas).

The non-glial cel tumors are a large heterogenous group of tumors

1. meningioma is the most common.
2. Schwannoma
3. Pituitary

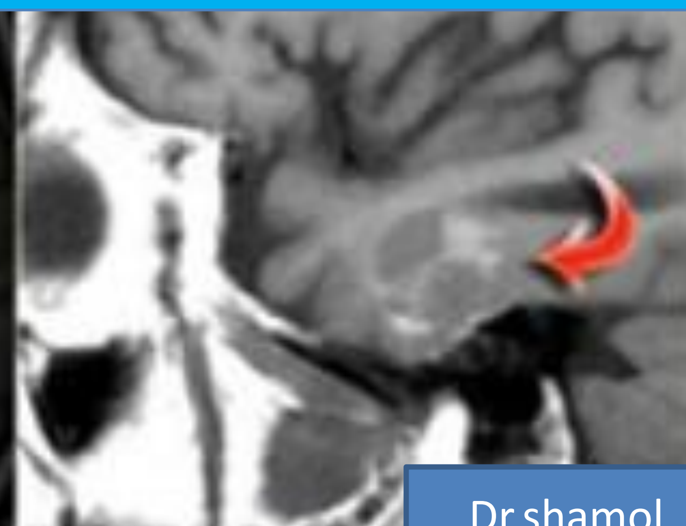


Most tumors have a low or intermediate signal intensity on T1WI. Except On the left
Calcifications are mostly dark on T1WI, but depending on the matrix of the calcifications they can sometimes be bright on T1.
Especially on gradient echo images slow flow can be seen as bright signal on T1WI and should not be confused with enhancement.
This is particularly pronounced on gradient echo images.
If you only do an enhanced scan, remember that high signal is not always enhancement.



hemorrhage in a pituitary macroadenoma.

hemorrhage in glioblastoma multiforme,



metastasis of a melanoma.

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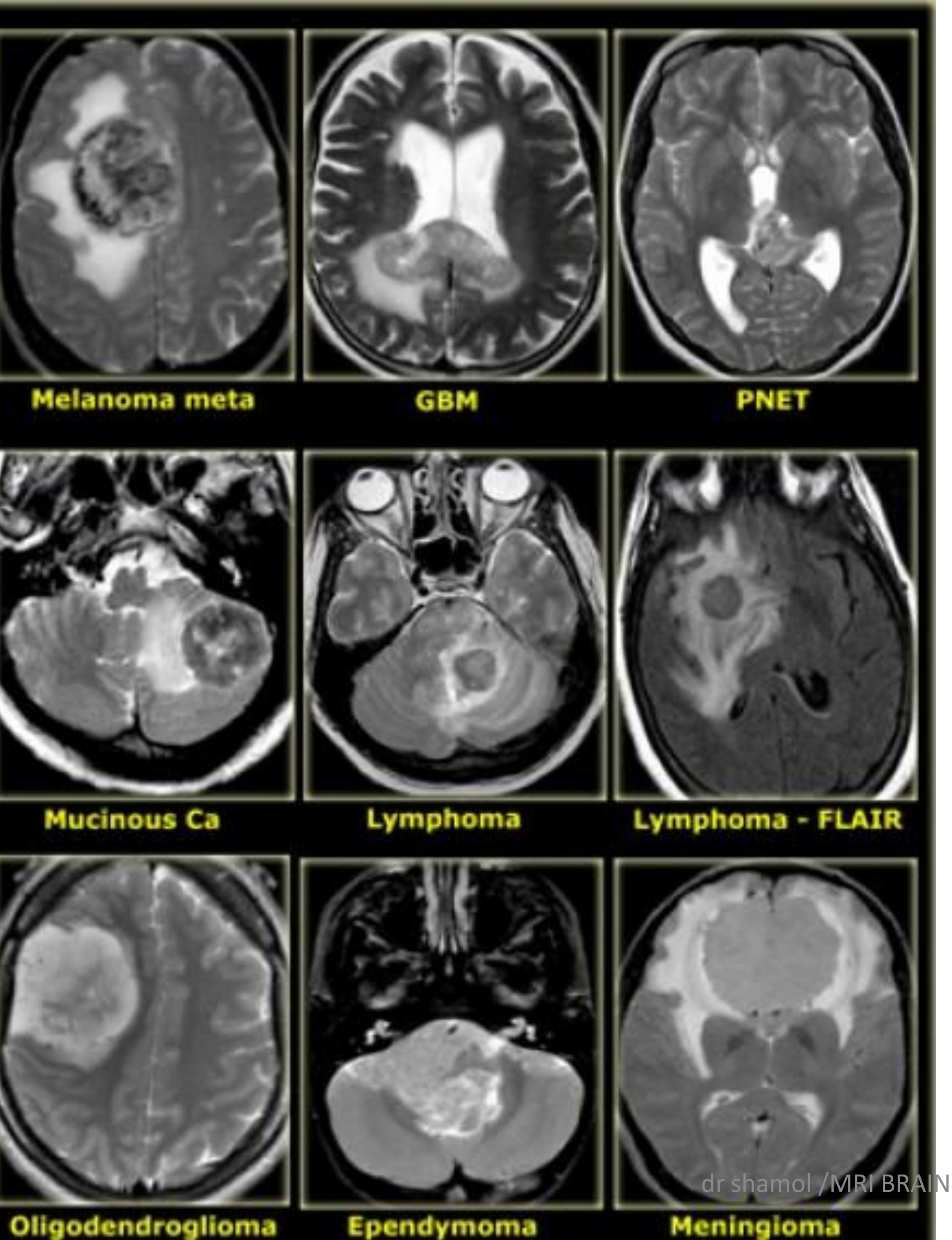
Low Intensity in Tumors on T2

Hypercellularity	→ Lymphoma, Meningioma, PNET Germinoma, GBM, Oligodendroglioma Mucinous-adeno metastases (GI, Lung, Breast, GU)
Calcification	→ see calcification
Blood	→ Old hemorrhage in tumor or vascular malformation
Protein	→ Colloid cyst
Melanin	→ Melanoma metastases
Flow-void	→ Heangioblastoma, Vascular malformation

Most tumors will be bright on T2WI due to a high water content.
When tumors have a low water content they are very dense and hypercellular and the cells have a high nuclear-cytoplasmasmic ratio.
These tumors will be dark on T2WI.
The classic examples are CNS lymphoma and PNET (also hyperdense on CT).
Calcifications are mostly dark on T2WI.
The differential diagnosis of calcified tumors was discussed above.
Paramagnetic effects cause a signal drop and are seen in tumors that contain hemosiderin.
Proteinaceous material can be dark on T2 depending on the content of the protein itself.
A classic example of this is the colloid cyst.
Flow voids are also dark on T2 and indicate the presence of vessels or flow within a lesion.
This is seen in tumors that contain a lot of vessels like hemangioblastomas, but also in non-tumorous lesions like vascular malformations.



- On the left some examples of tumors with a low signal intensity on T2WI.
- Melanoma metastases have a low SI on T2WI as a result of the melanin.
- GBM can have a low SI on T2WI because sometimes they have a high nuclear-cytoplasmic ratio. Most GBM's, however, are hyperintense on T2WI.
- PNET typically has a high nuclear-cytoplasmic ratio. PNET is mostly located in the region of the 4th ventricle, but another, less common, location is in the region of the pineal gland.
- Mucinous metastases can have a low SI on T2WI because they often contain calcifications..
- Meningiomas are mostly of intermediate signal.

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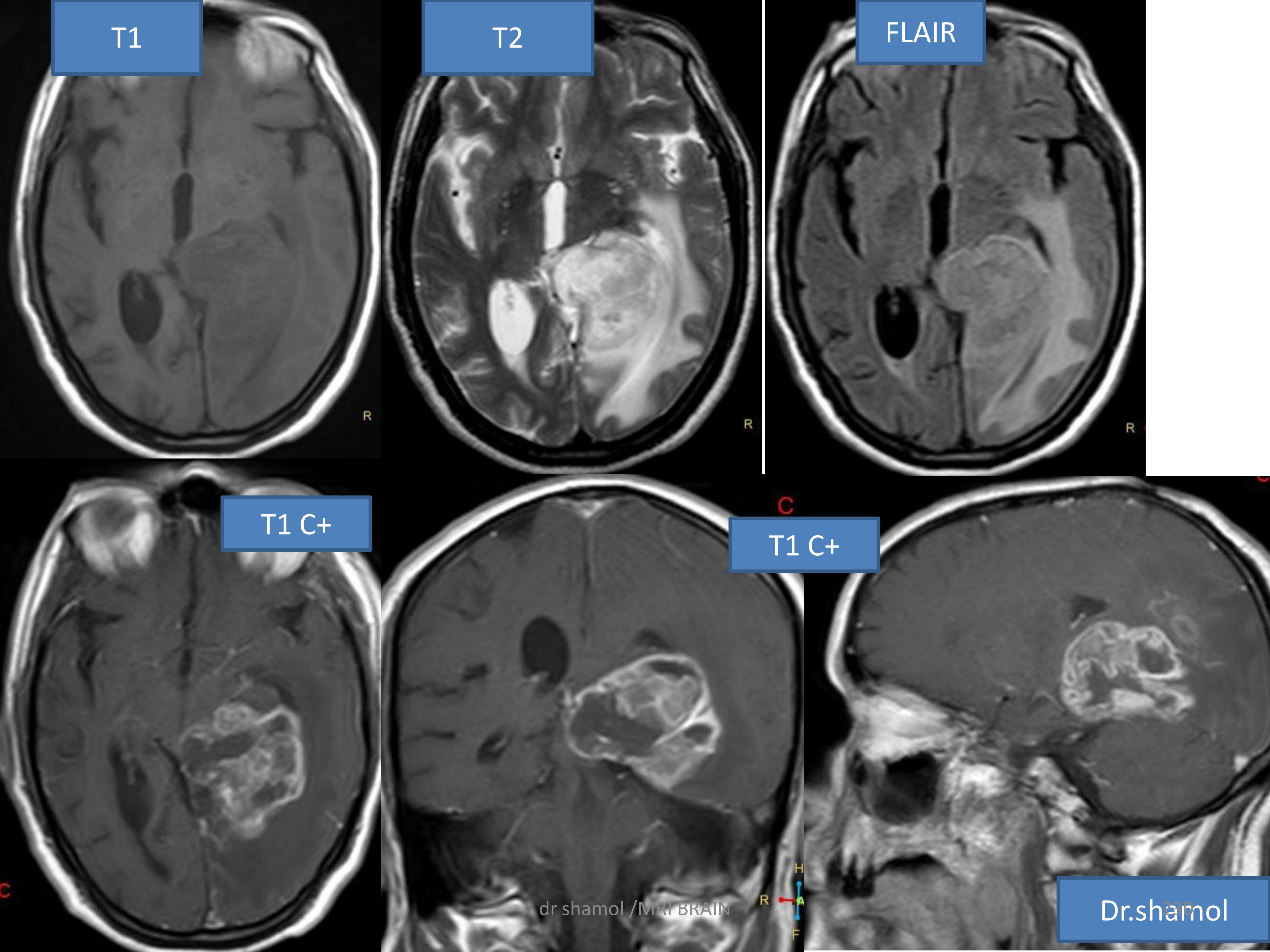
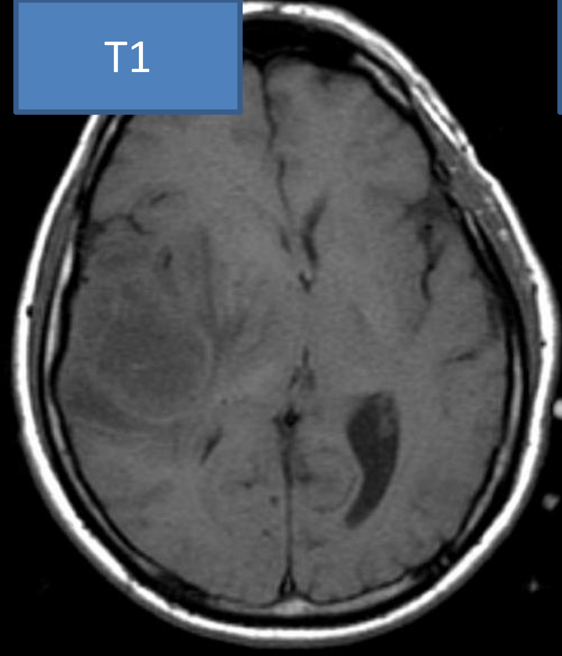
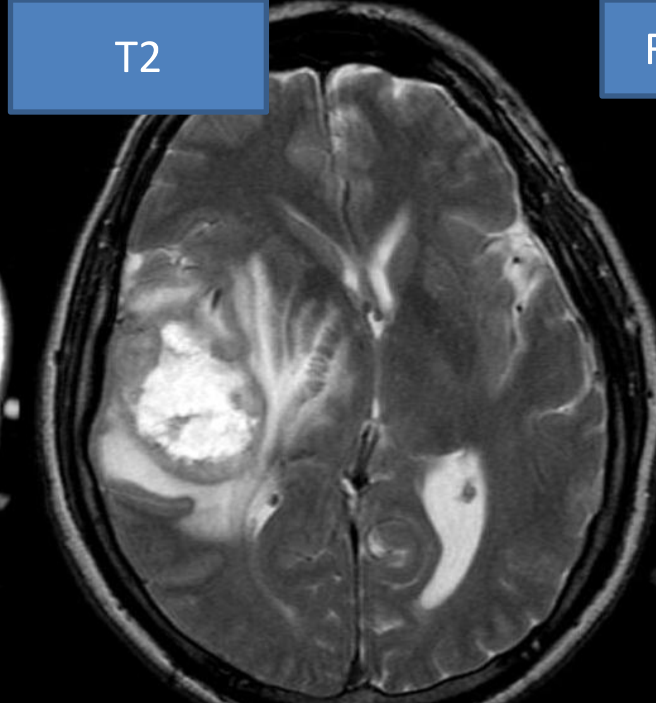
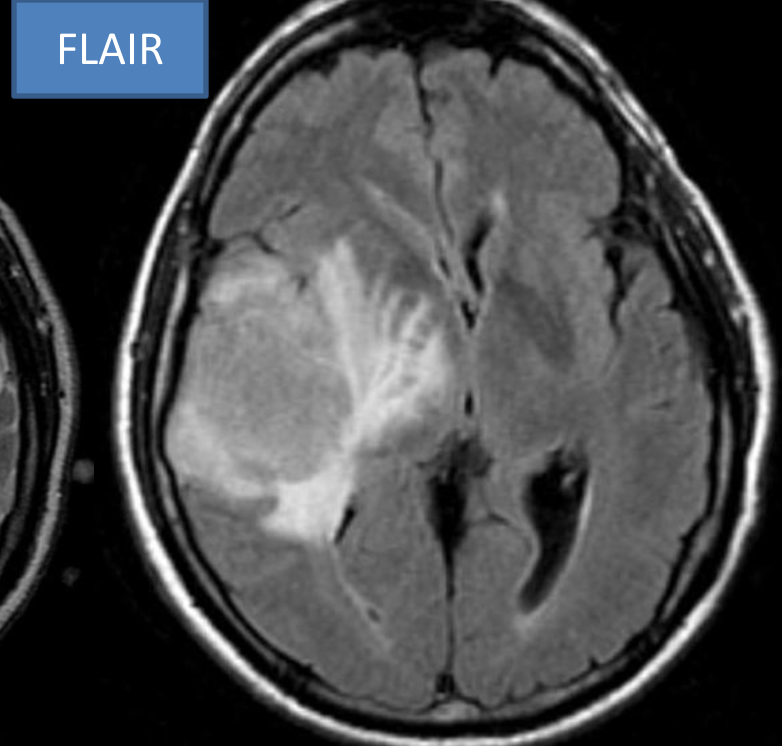
They can have a high SI on T2WI if they contain a lot of water.
They can have a low SI on T2WI if they are very dense and hypercellular or when they contain calcifications.

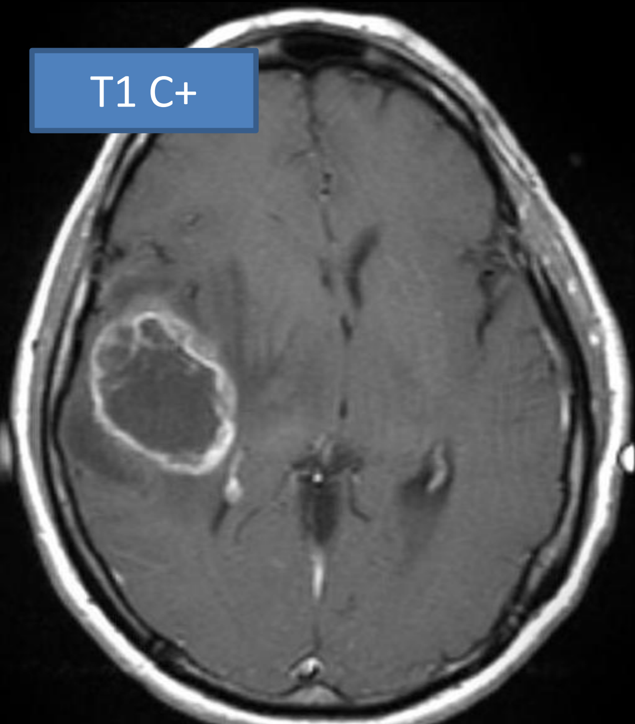
Glioblastoma

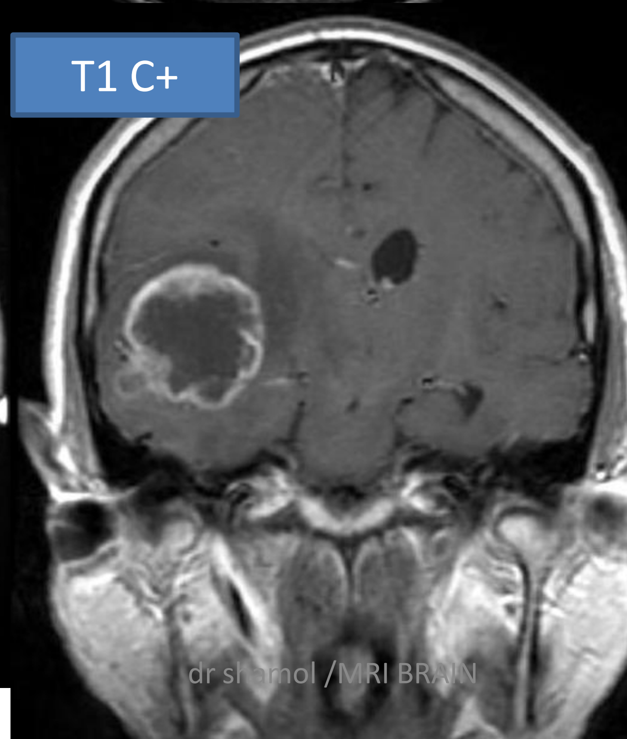

T1
T2
FLAIR
R
R
R
T1 C+
C
T1 C+
H
R
F
dr shamol /MRI BRAIN
Dr.shamol
230











It exerts significant mass effect and is surrounded by vasogenic oedema. The mass itself is heterogeneously low on T1, and demonstrates a very high T2 signal region centrally, suggestive of a necrotic centre.
Following administration of contrast, the margins enhance irregularly.

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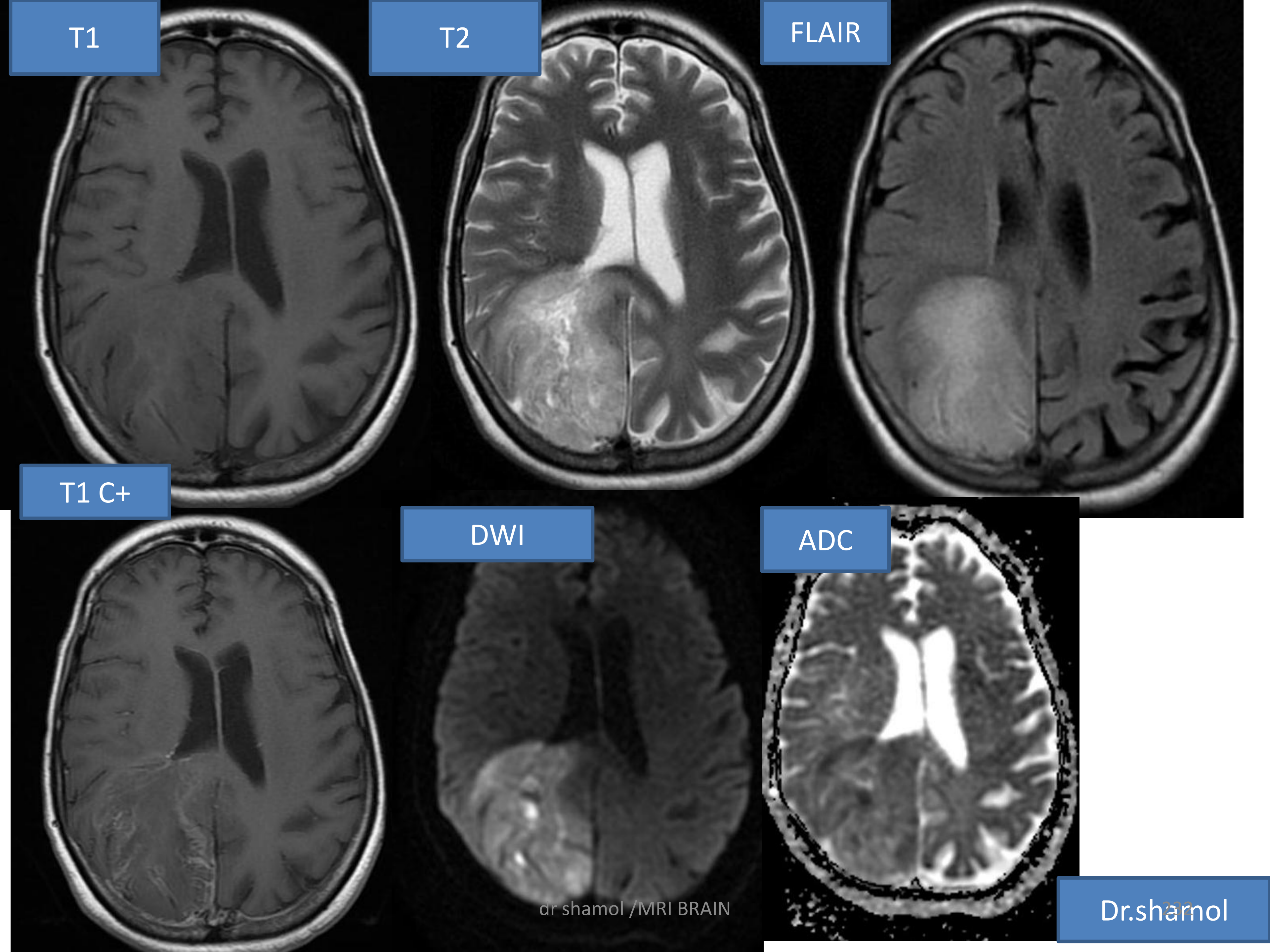
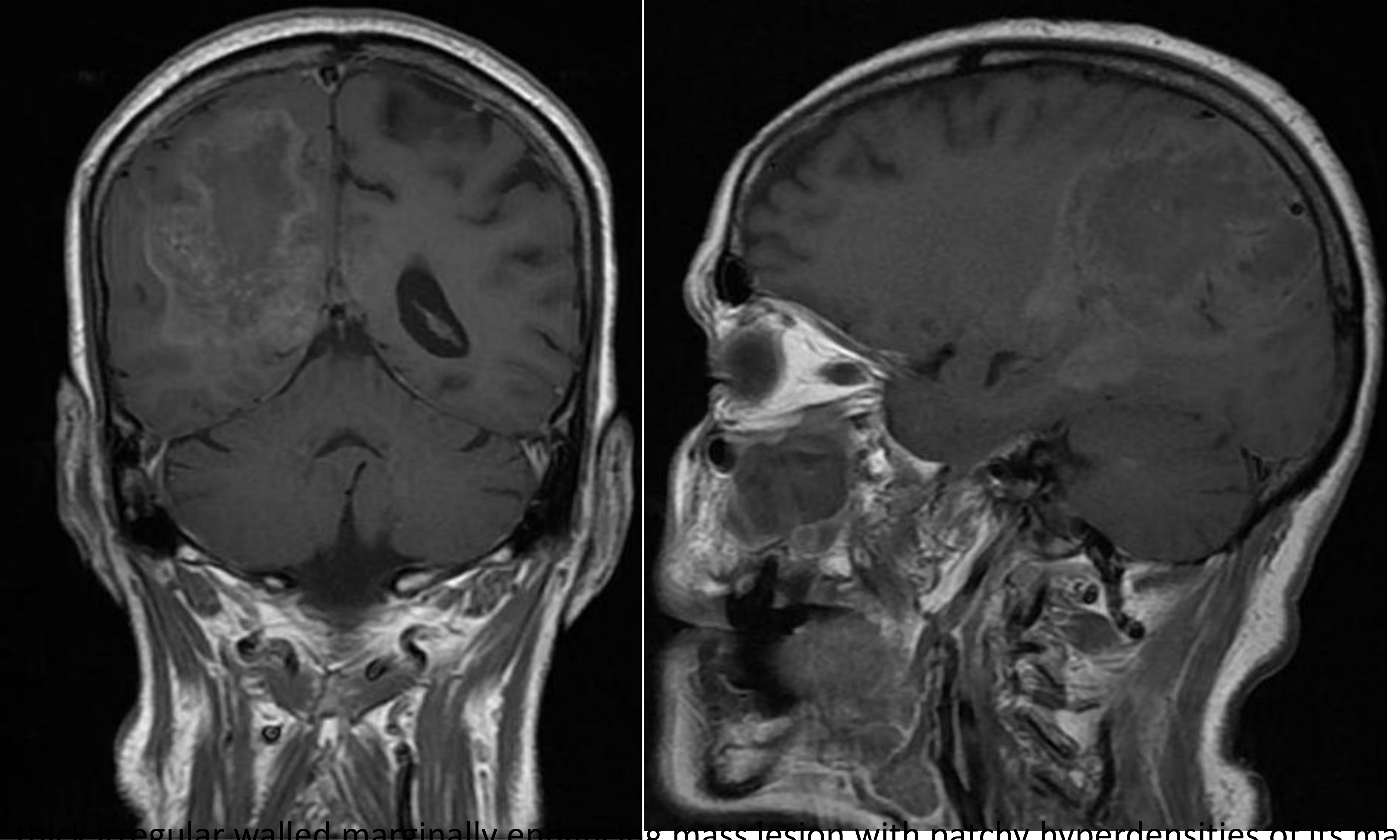
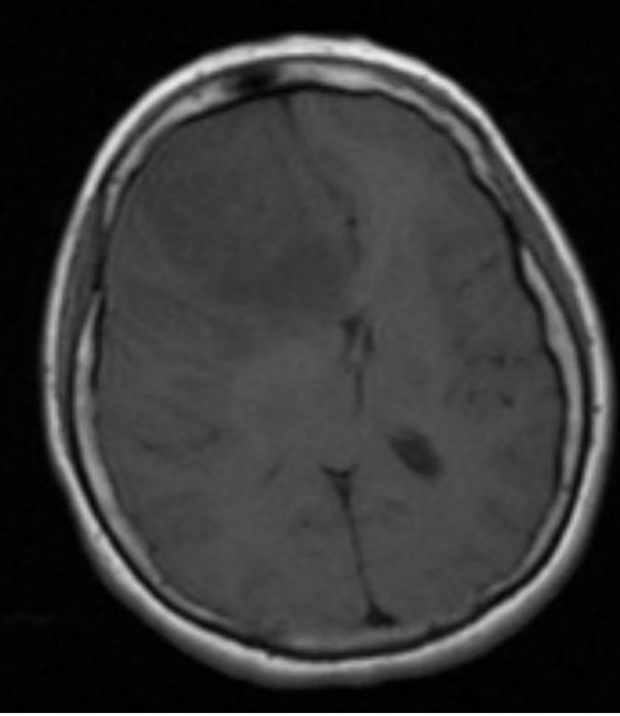
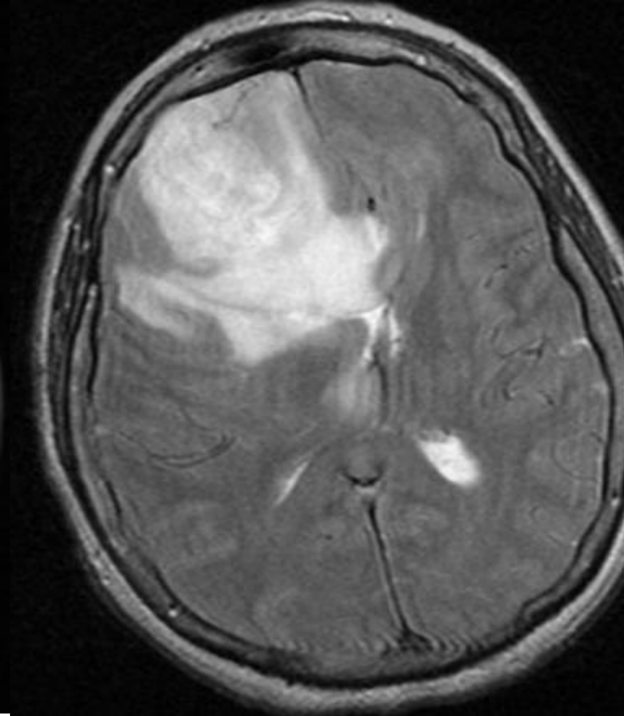
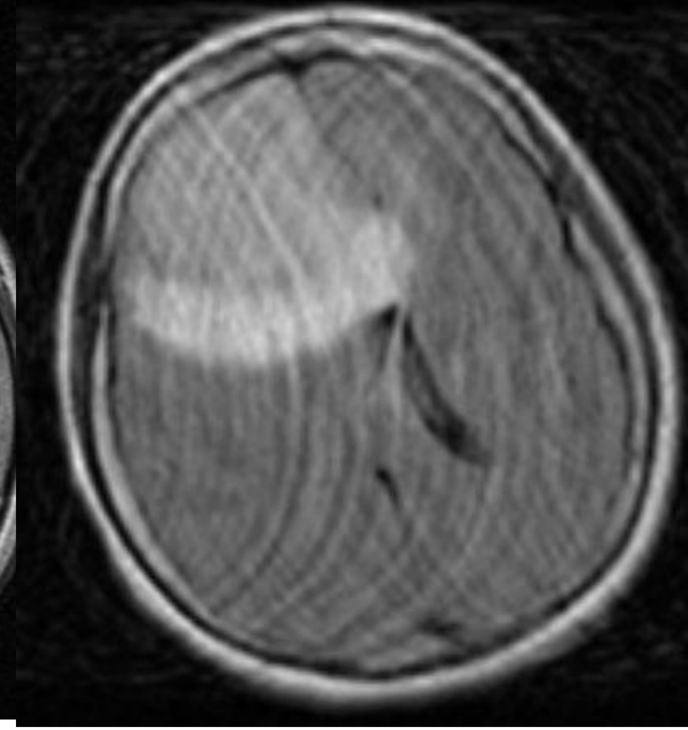
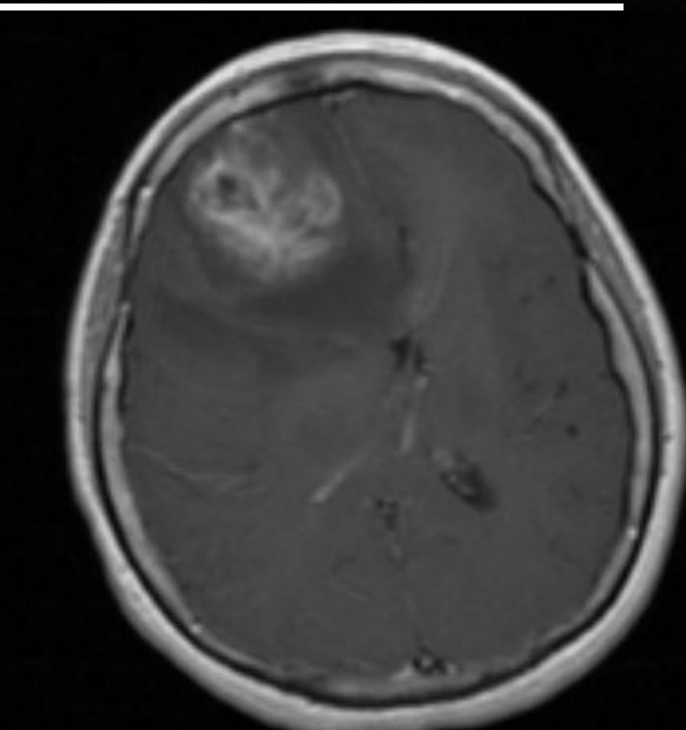
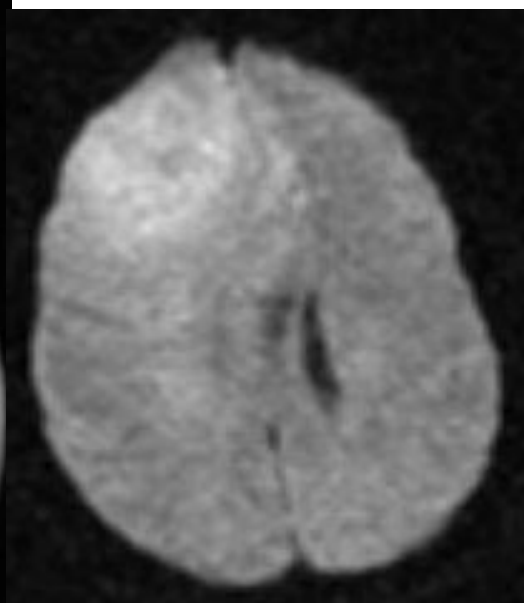
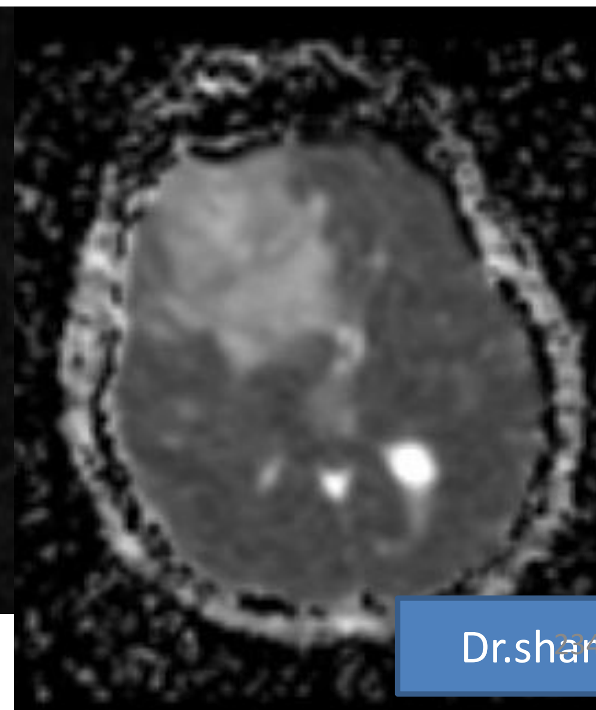

T1
T2
FLAIR
T1 C+
DWI
ADC
Dr.shamol



thick irregular walled marginally enhancing mass lesion with patchy hyperdensities of its margin and central necrosis is seen. The mass demonstrates predominantly hypointense T1 signal with patchy hyperintensities (met Hb signal) and hyperintense T2/Flair signal with restricted diffusion predominantly of its thick irregular margin The mass lesion is seen surrounded by vasogenic brain oedema with compression of the right lateral ventricle's body and occipital horn as well as slight contralateral midline shift

LOW grade glioma
ASTROCYTOMA

T1

isointense to hypointense compared to white matter

T2/FLAIR

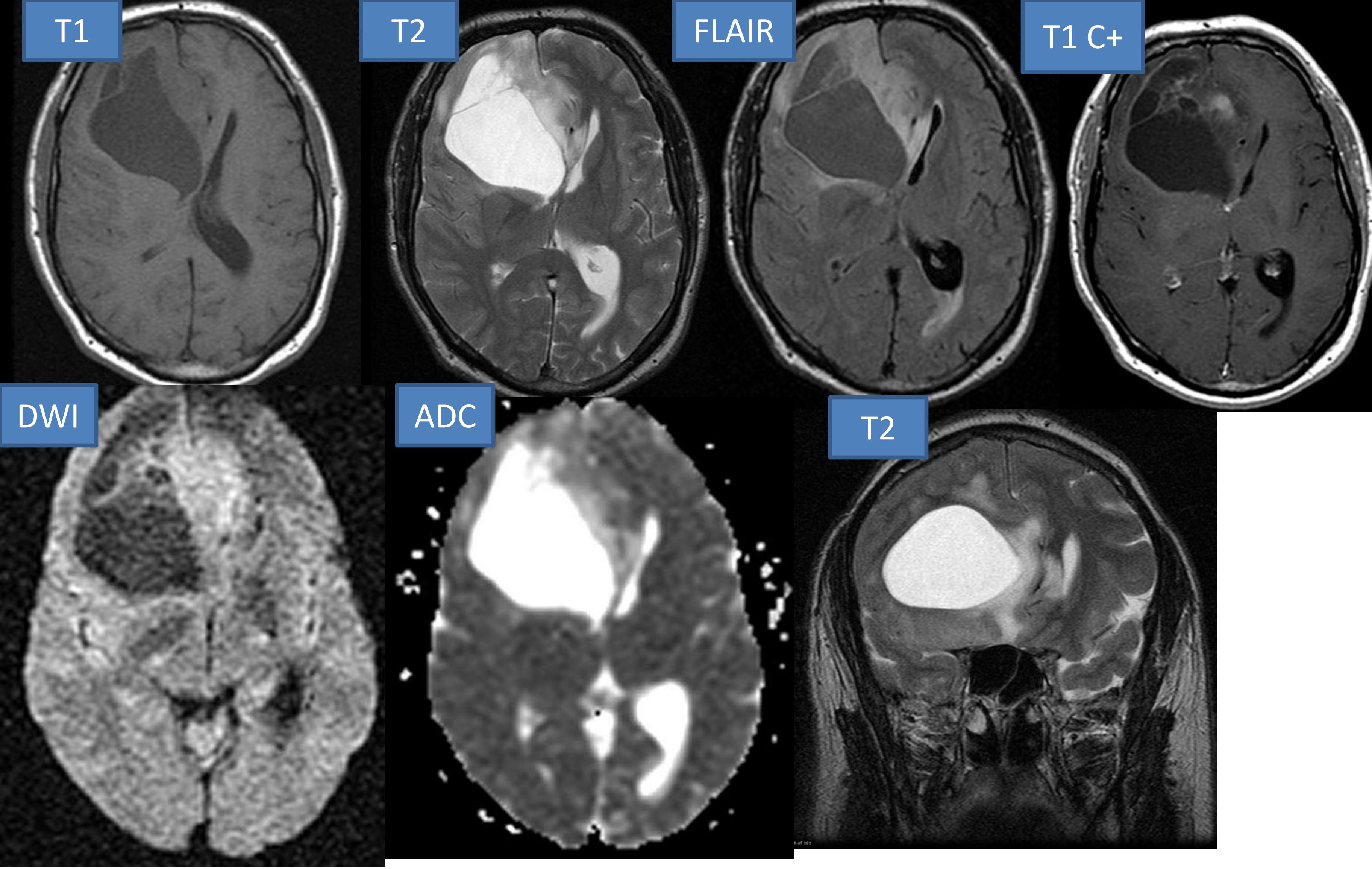
hyperintense signal

DWI/ADC

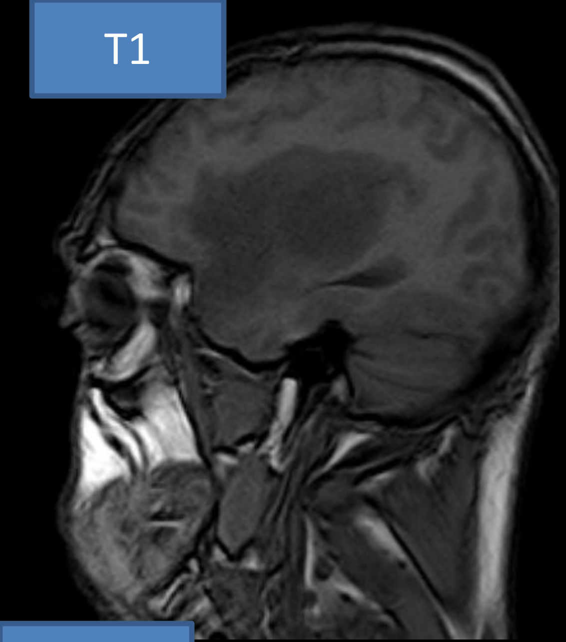
typically has facilitated diffusion

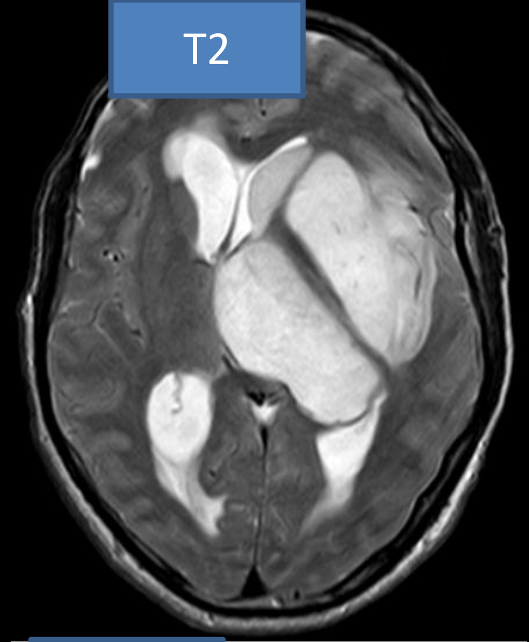
T1 C+ (Gd)

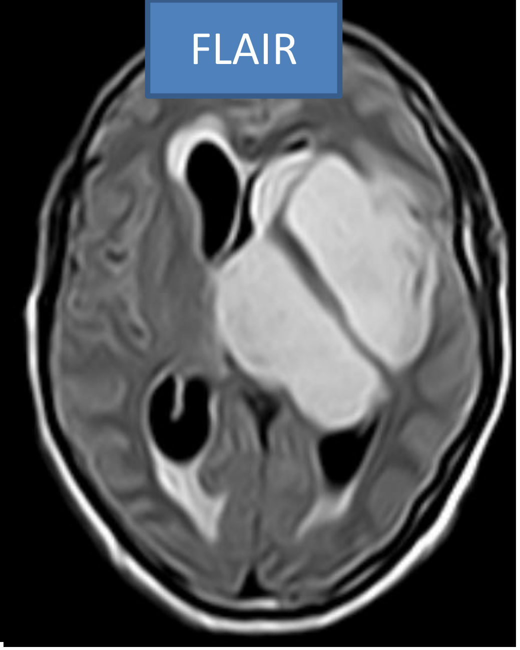
no enhancement is often the rule but small ill-defined areas of enhancement are not rare; however, when enhancement is seen it should be considered as a warning sign for progression to a higher grade

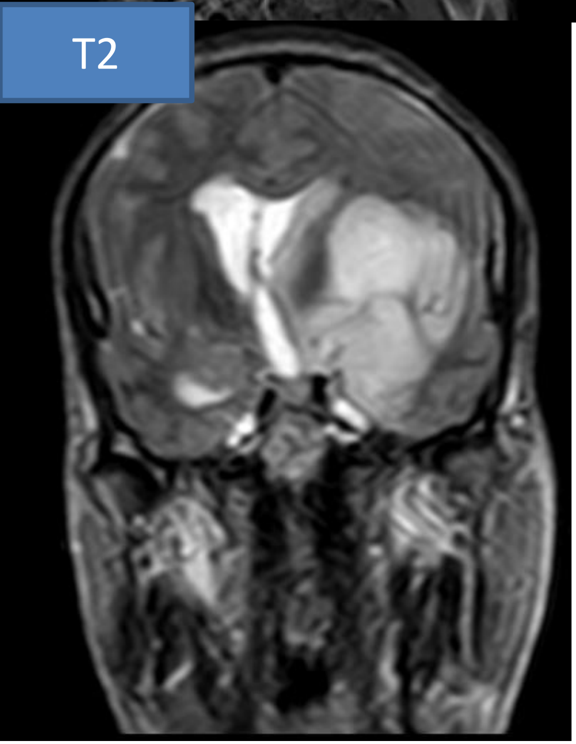


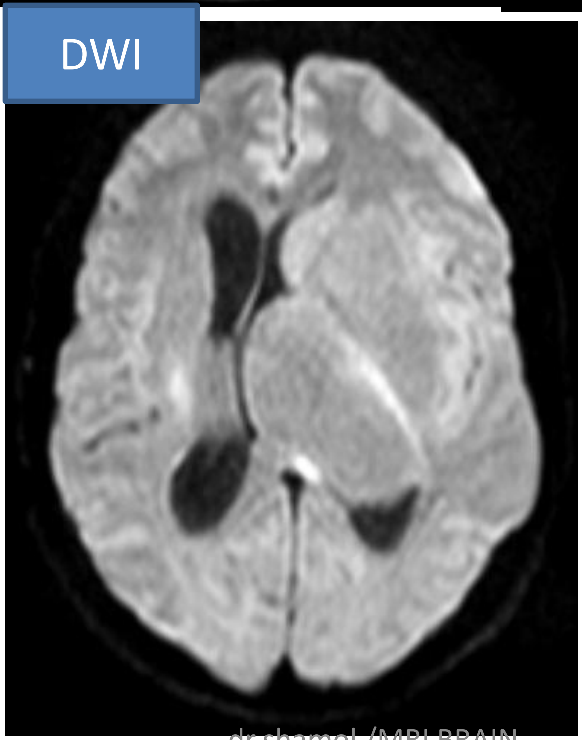
- ❖low / hypo intens in T1,
- ❖high intens in T2/FLAIR
- ❖ post contrast no enhancement
- ❖ DWI reveals (black) No diffusion restriction









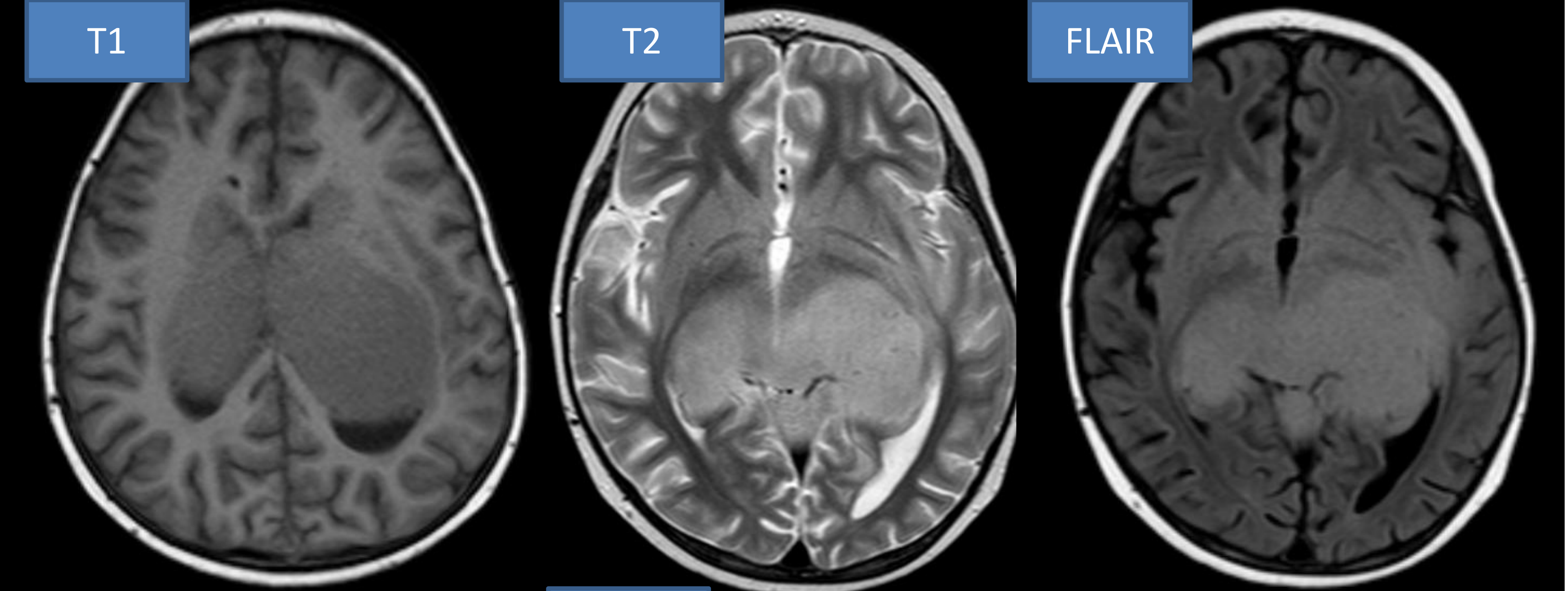


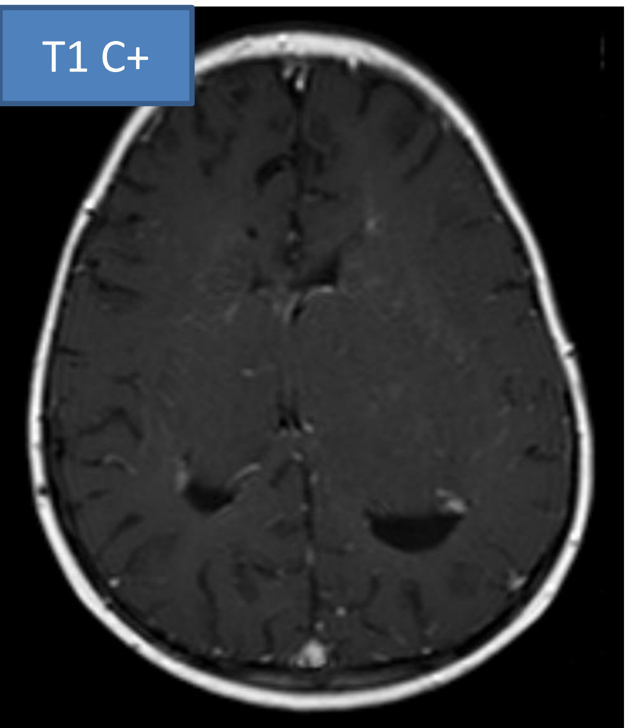
Diffuse involvement of the left insular cortex, medial part of left temporal lobe, left lentiform and caudate nuclei as well as left thalamus

❖low / hypo intens in T1,

❖high intens in T2/FLAIR

❖ post contrast no enhancemen





on T1 –iso-intens
on T2 and FLAIR--bright signal /hyper intens
on T1 C+ – no enhancement on post contrast study.
DWI reveals an area of faint diffusion restriction on the most superior part of left side thalamic lesion

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Meningioma

T1

usually isointense to grey matter (60-90%)

hypointense to grey matter (10-40%): particularly fibrous, psammomatous variants

T1 C+ (Gd):

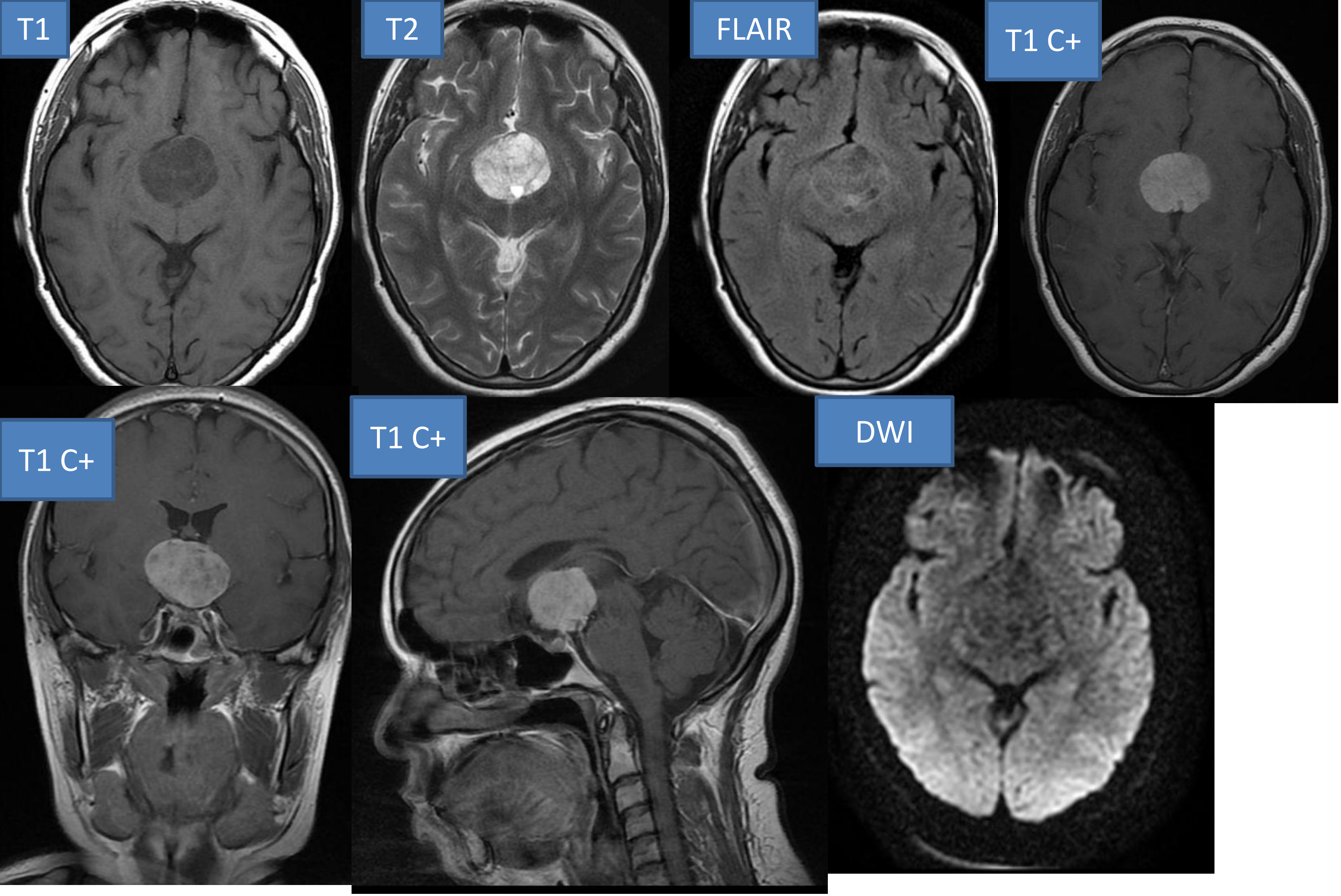
usually intense and homogeneous enhancement

T2

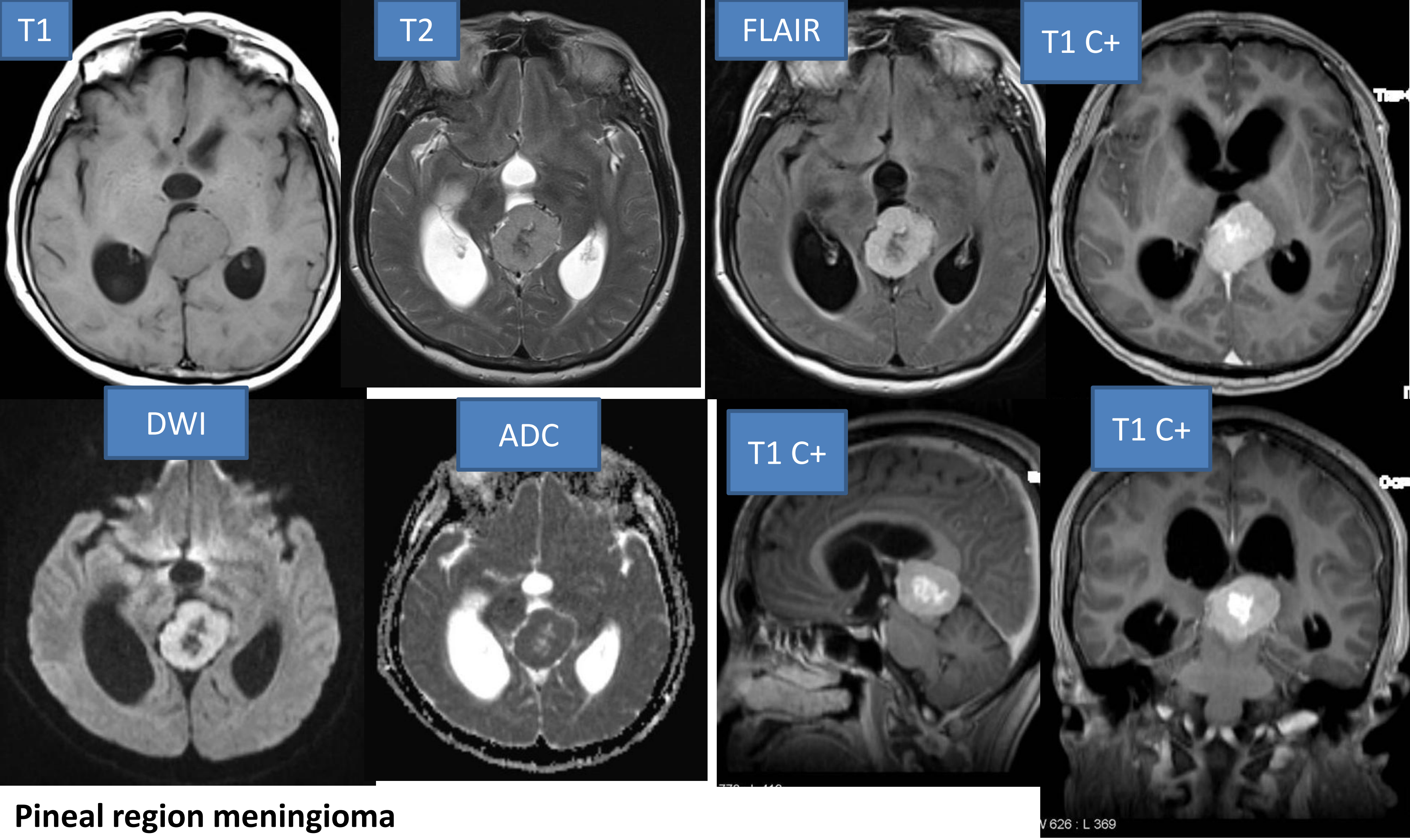
usually isointense to grey matter (~50%)

hyperintense to grey matter (35-40%)

hypointense to grey matter (10-15%):



Suprasellar meningioma

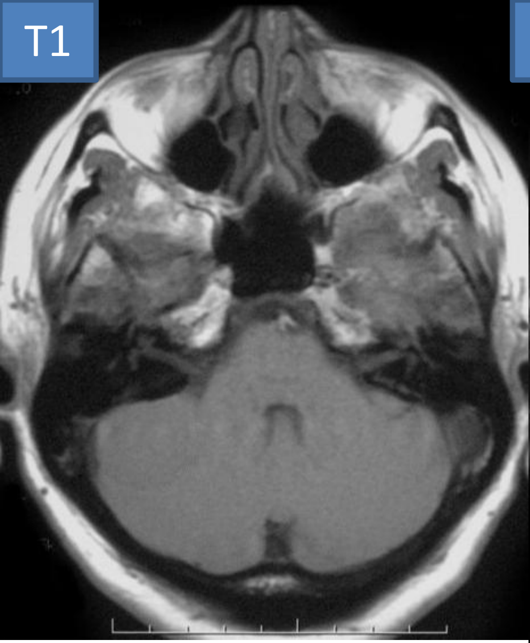


Pineal region meningioma

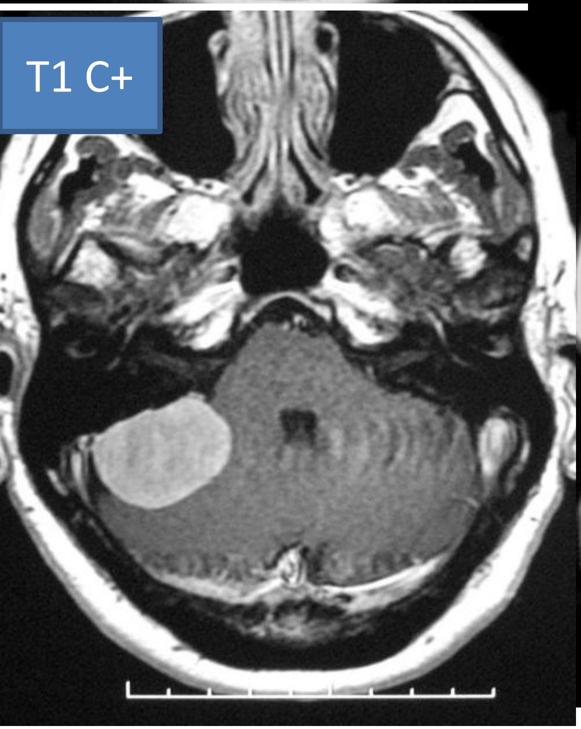
Isointense to grey matter on both T1 and T2

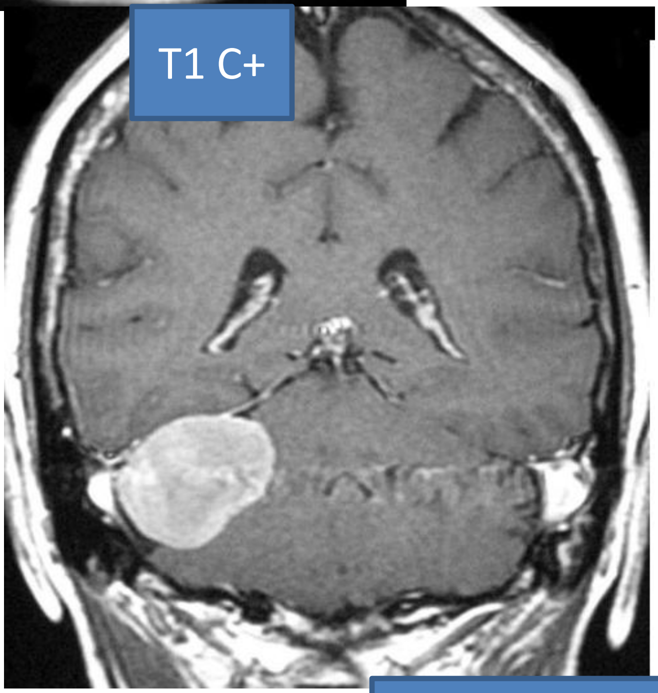
central region of lower T2 signal (possibly due to a vascular core).It demonstrates vivid contrast enhancement.

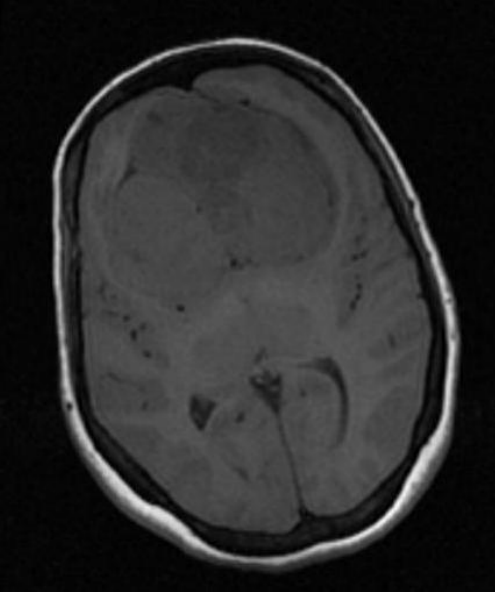
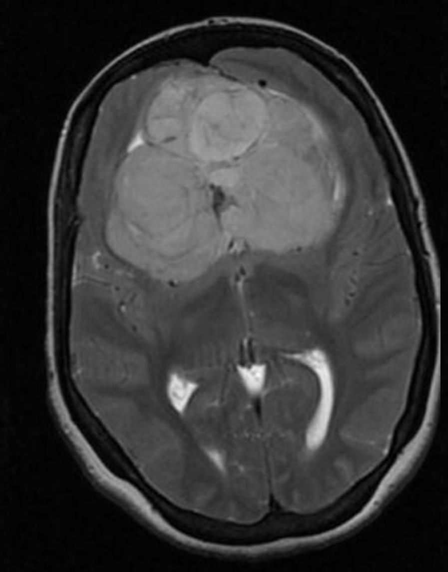
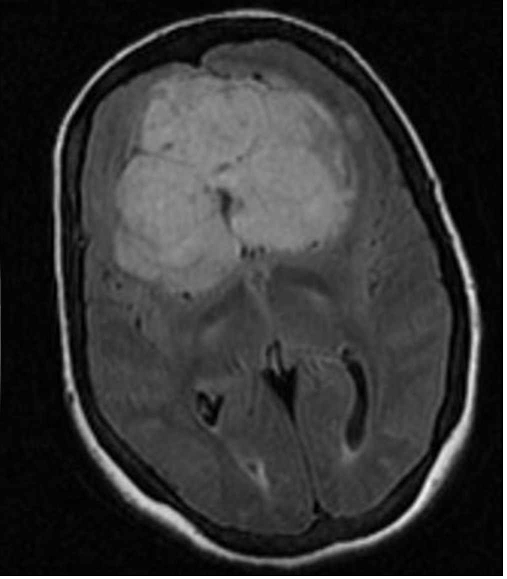
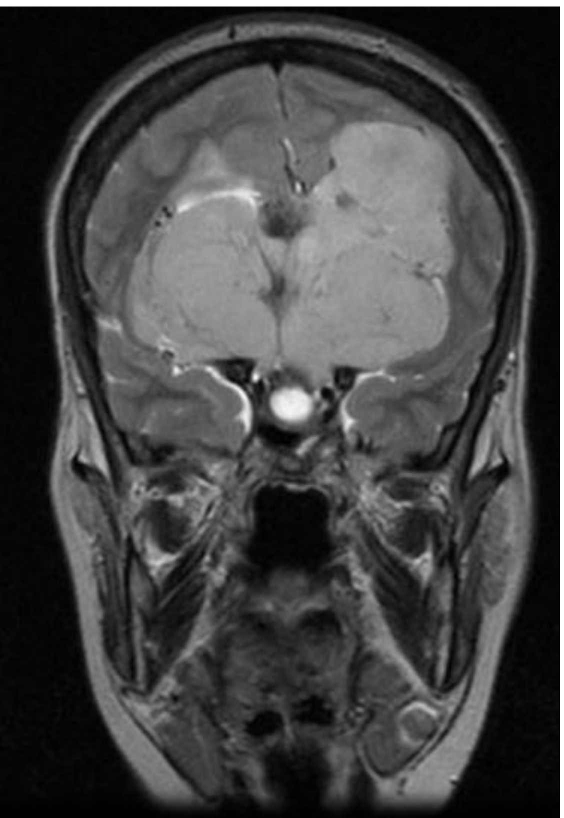
Restricted diffusion is seen throughout most of the mass, sparing the central regi

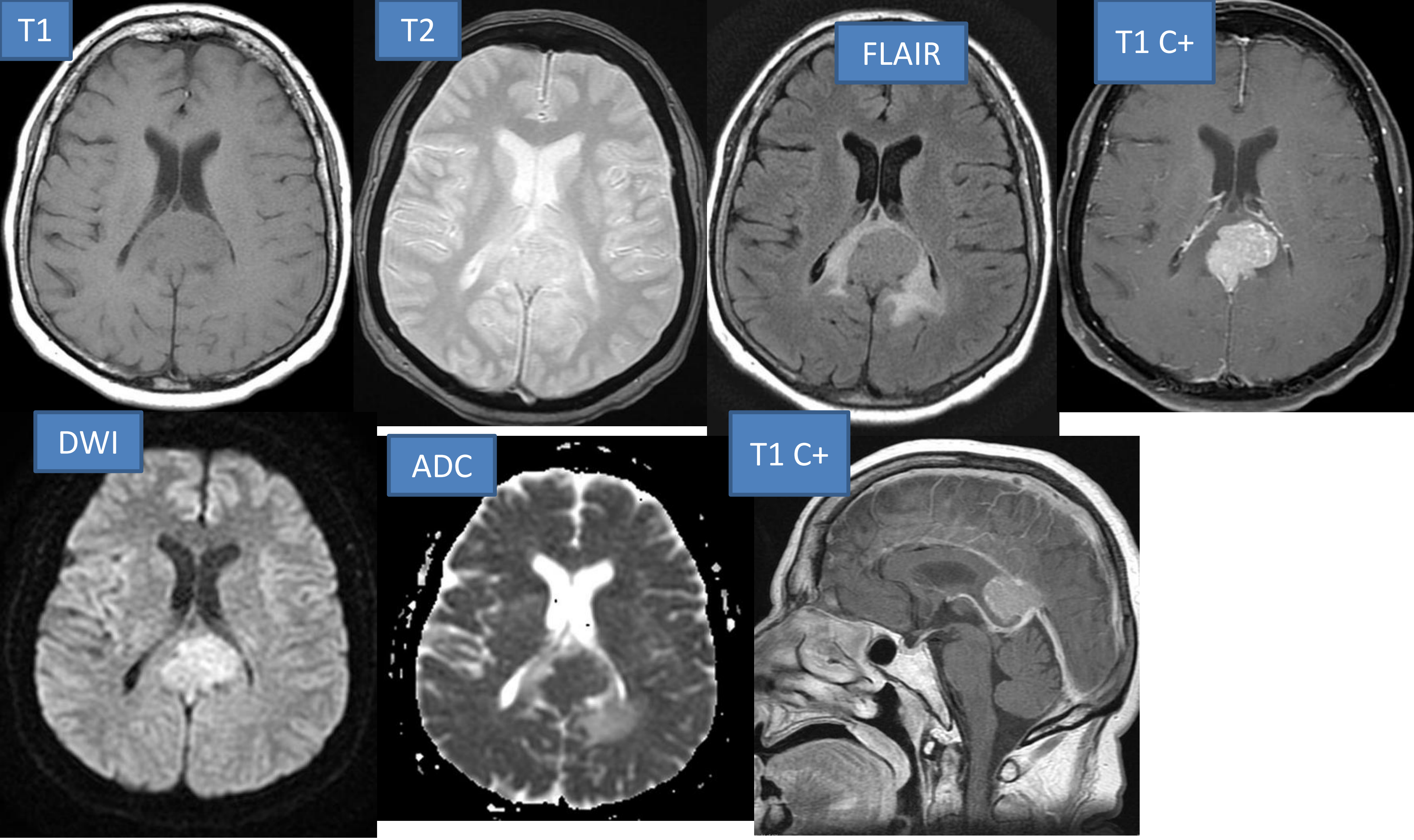


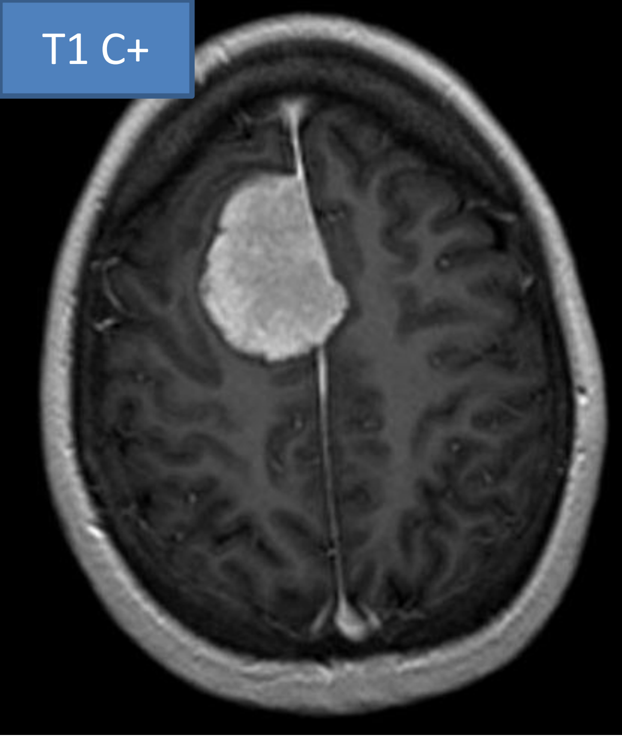
vividly enhancing extra-axial mass posterior to the internal acoustic meatus. It is isointense to adjacent cerebellum on both T1 and T2 weighted images.

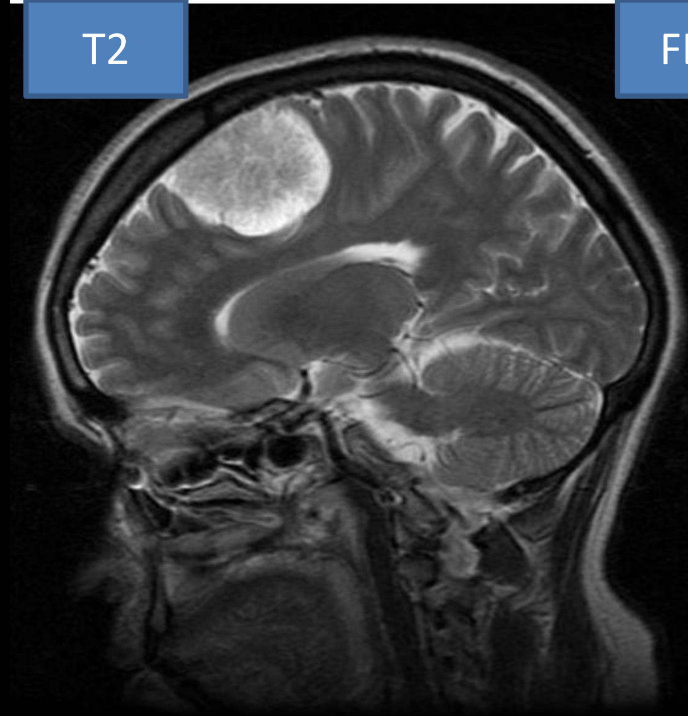


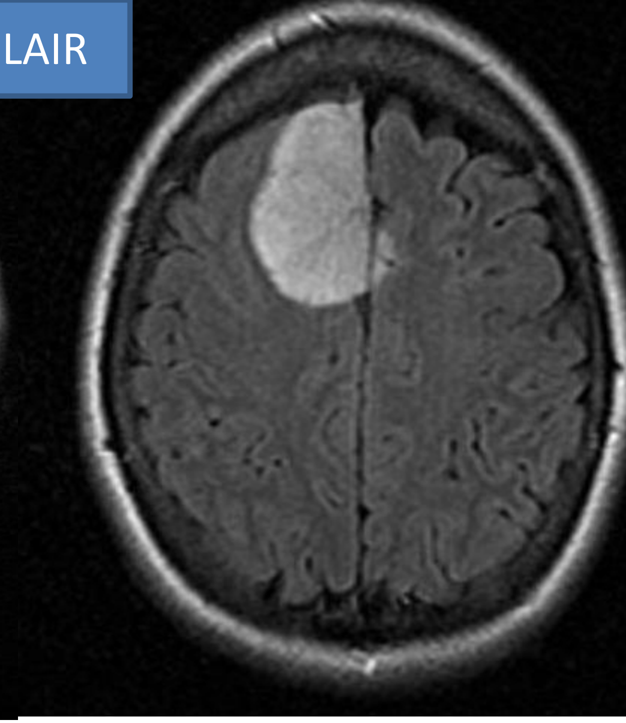



T1
T2
FLAIR
T1 C+
DWI
ADC
T1 C+

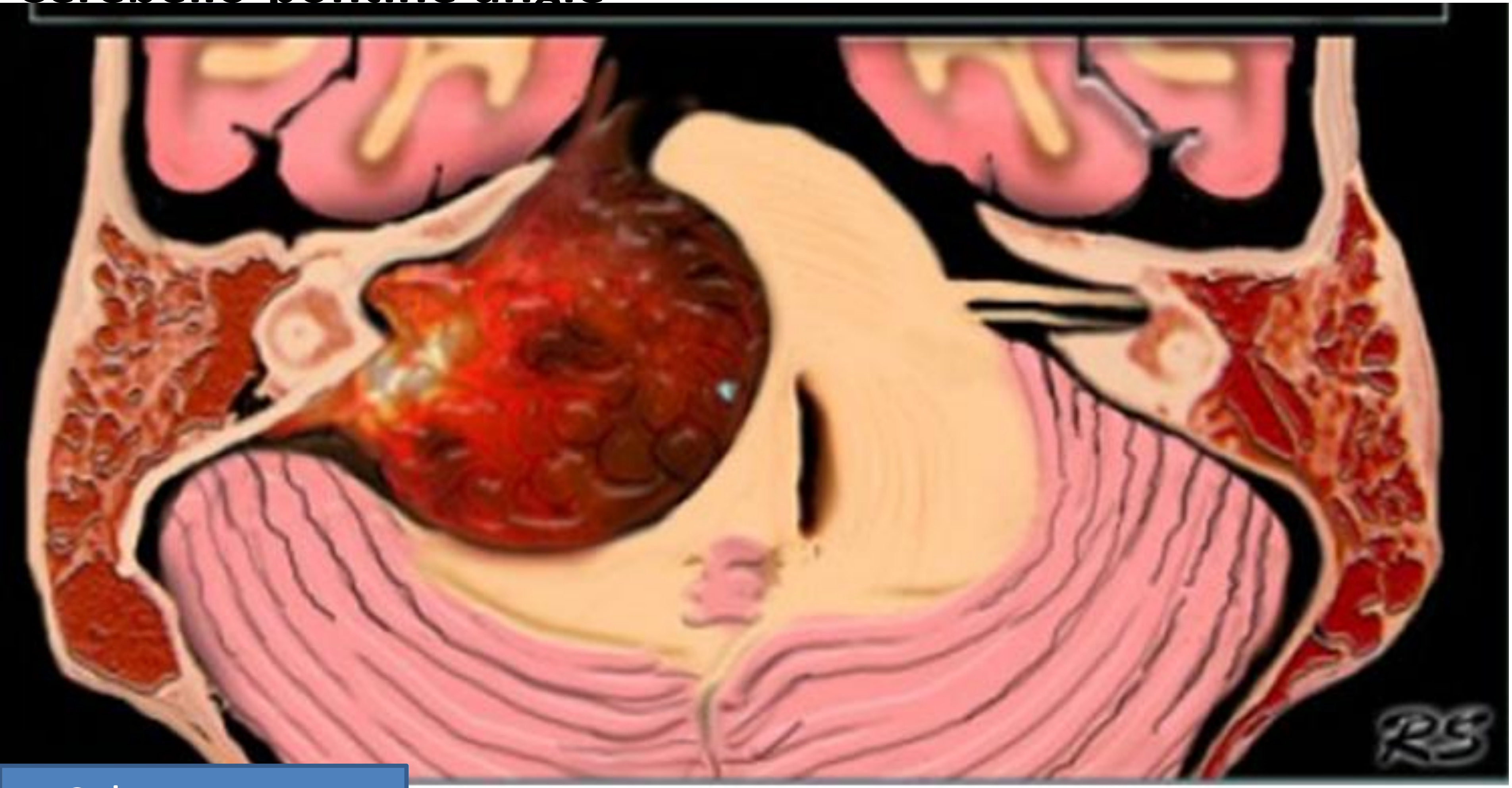






bright signal on FLAIR and T2 and
strong enhancement after contrast administration T1C+

Cerebello-pontine angle



Schwannoma
Meningionma
Epidermoid
Arachnoid cyst
Metastasis

schwannoma

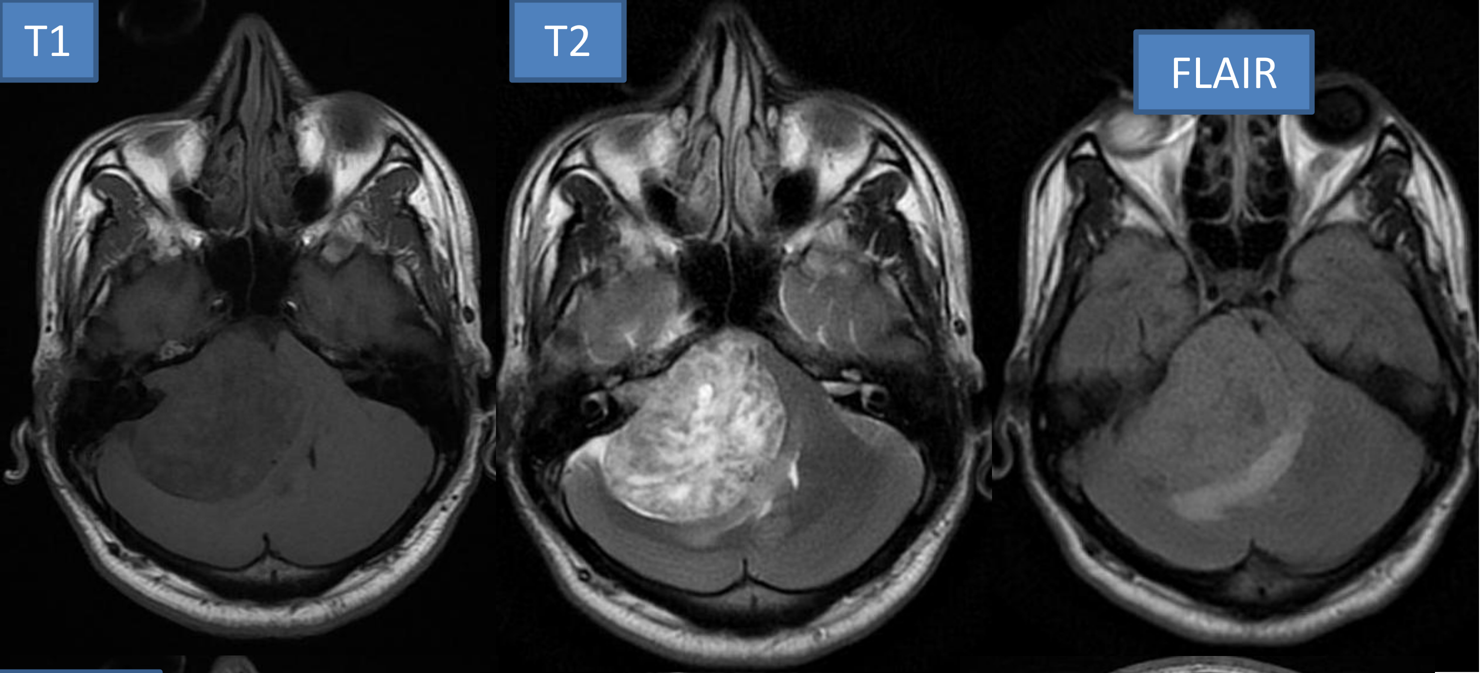
T1
- isointense to hypointense to brain
- low signal cystic areas, if present

T1 C+ (Gd)
- prominent enhancement
- heterogeneous in 70% of cases [1]

T2
- typically somewhat hyperintense to brain
- cystic areas are hyperintense

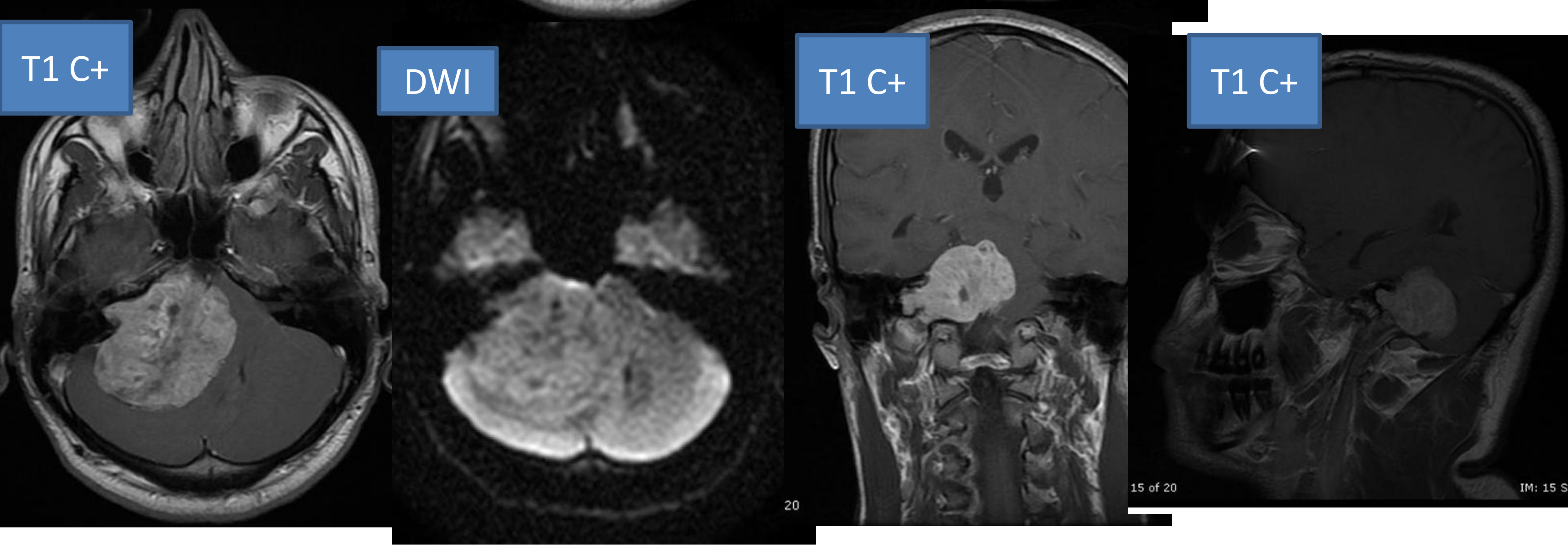
T2* (GE / SWI)
- haemosiderin staining may be encountered, particularly in larger tumours [2]

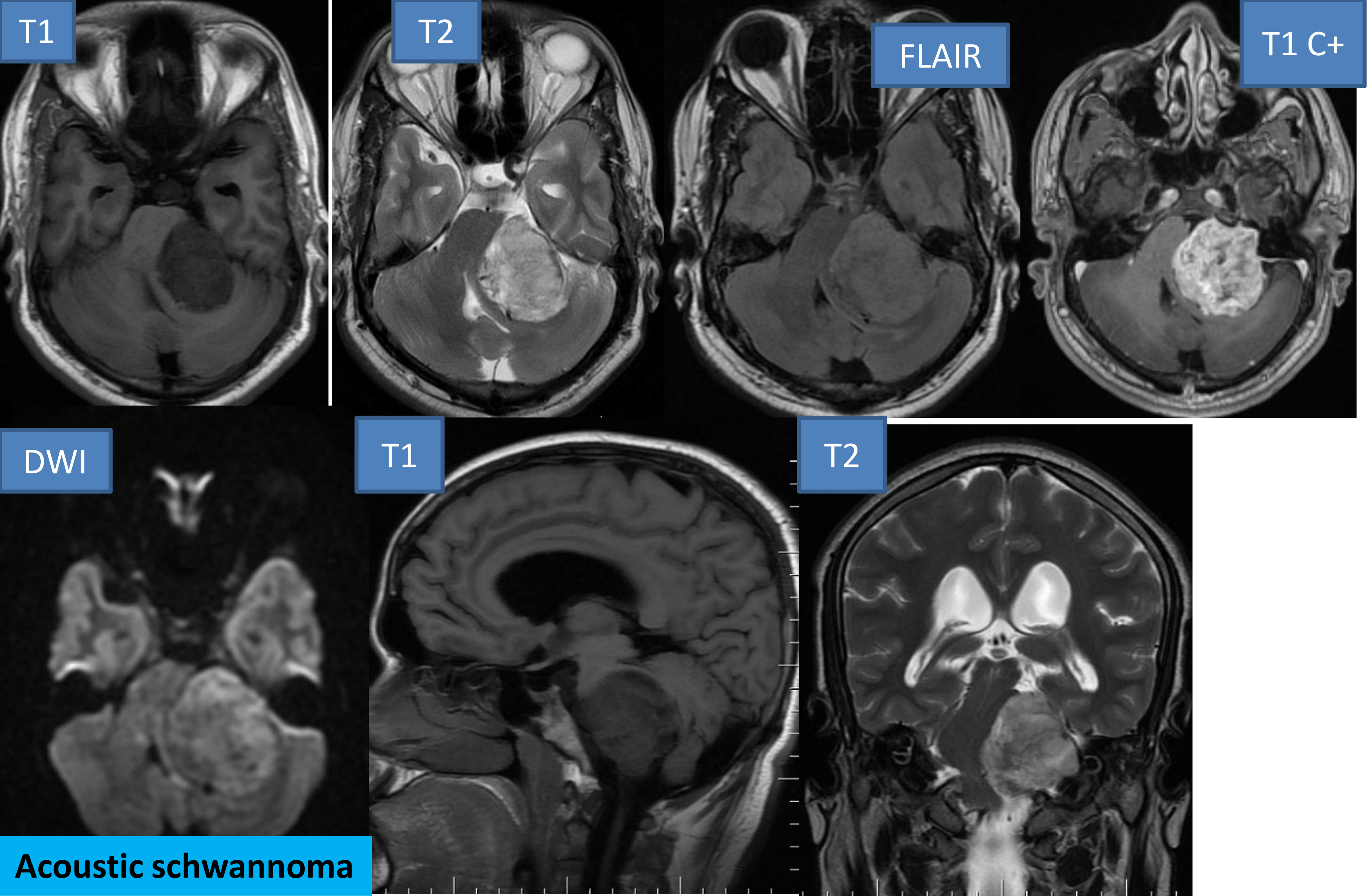
DWI/ADC
- often higher signal on both DWI and ADC (T2 shine through - not restricted diffusion)



Acoustic neuroma

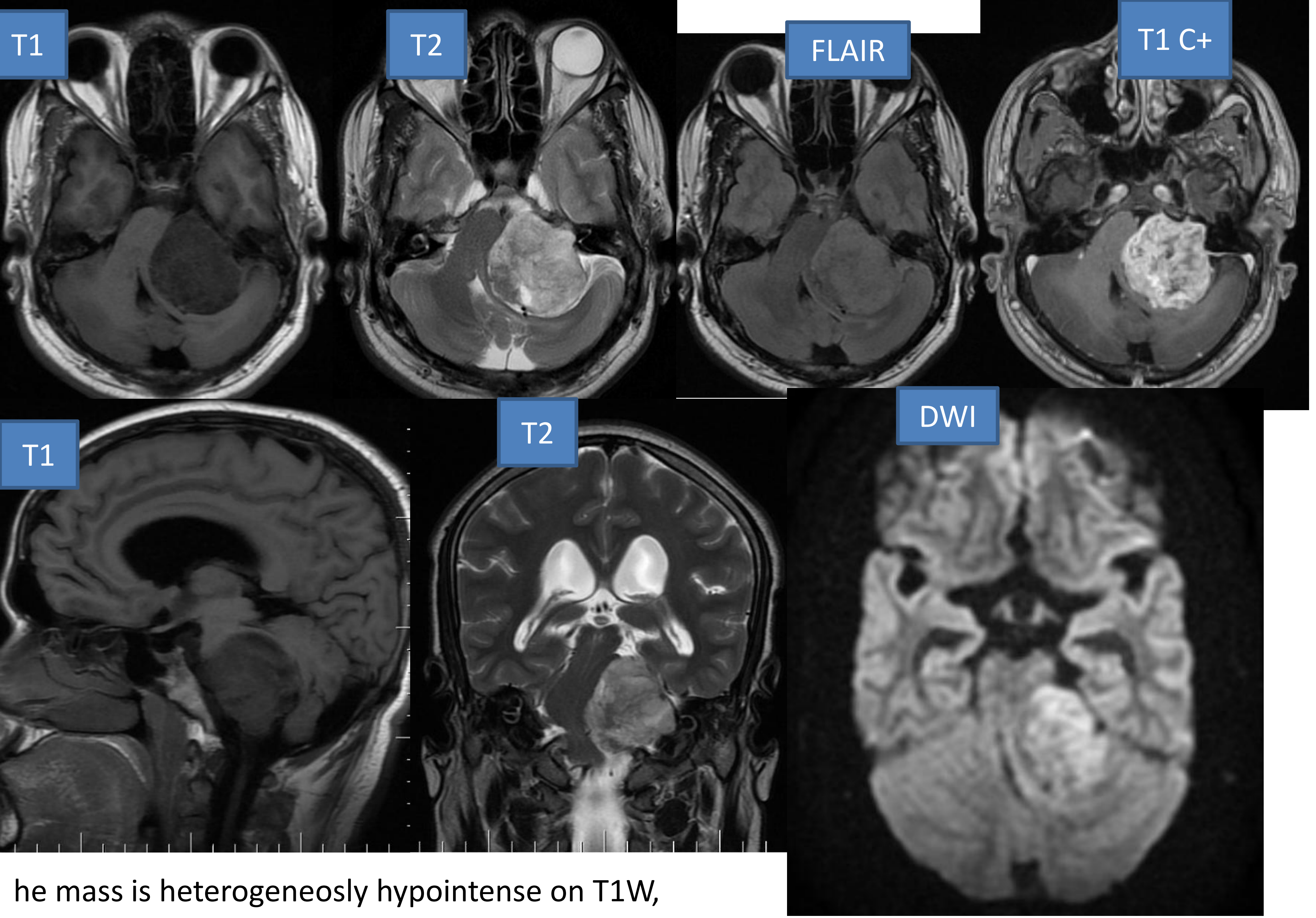
shows heterogenous hypointense T1 and hyperintense T2 signal with central necrosis. The lesion shows intense heterogenous post-contrast enhancement





Acoustic schwannoma

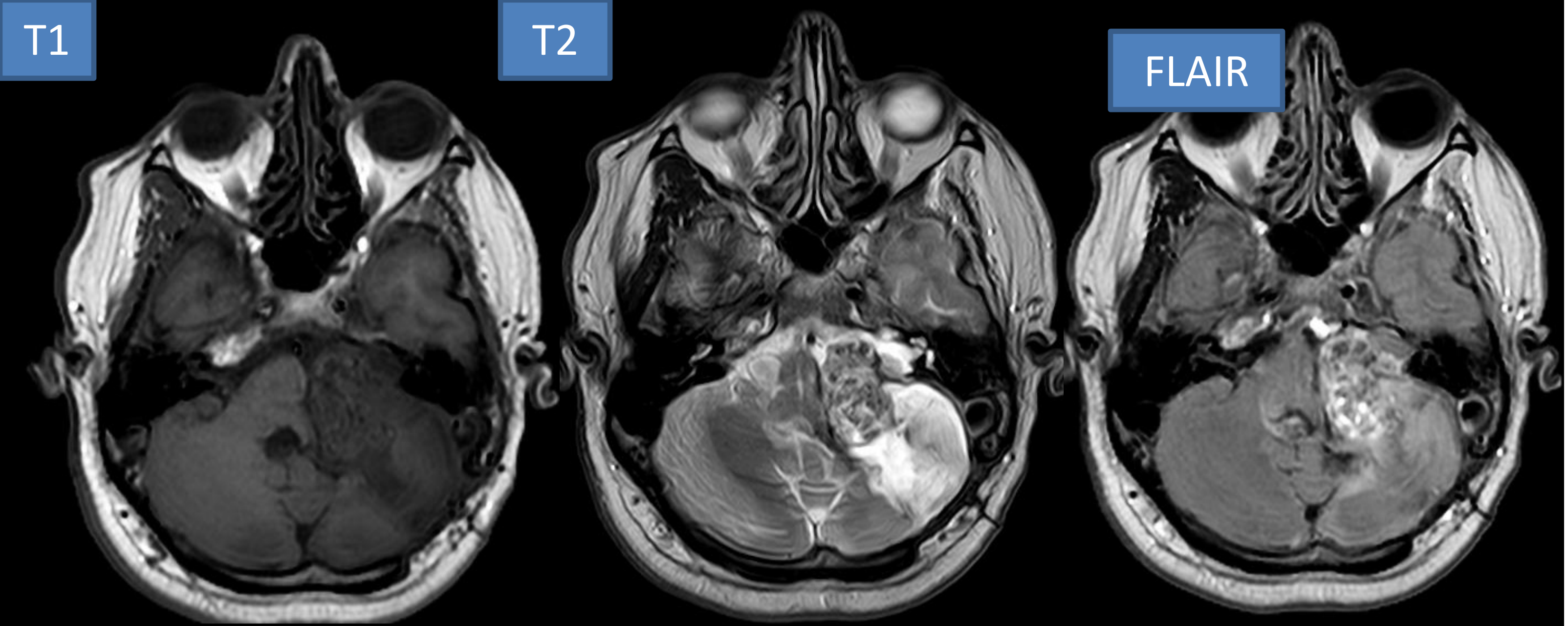
The mass is heterogeneosly hypointense on T1W, hyperintense on T2W and FLAIR sequences.
Few tiny foci of blooming on gradient echo sequence suggestive of haemorrhage.
Multiple non-enhancing cystic necrotic areas are seen within the tumour.

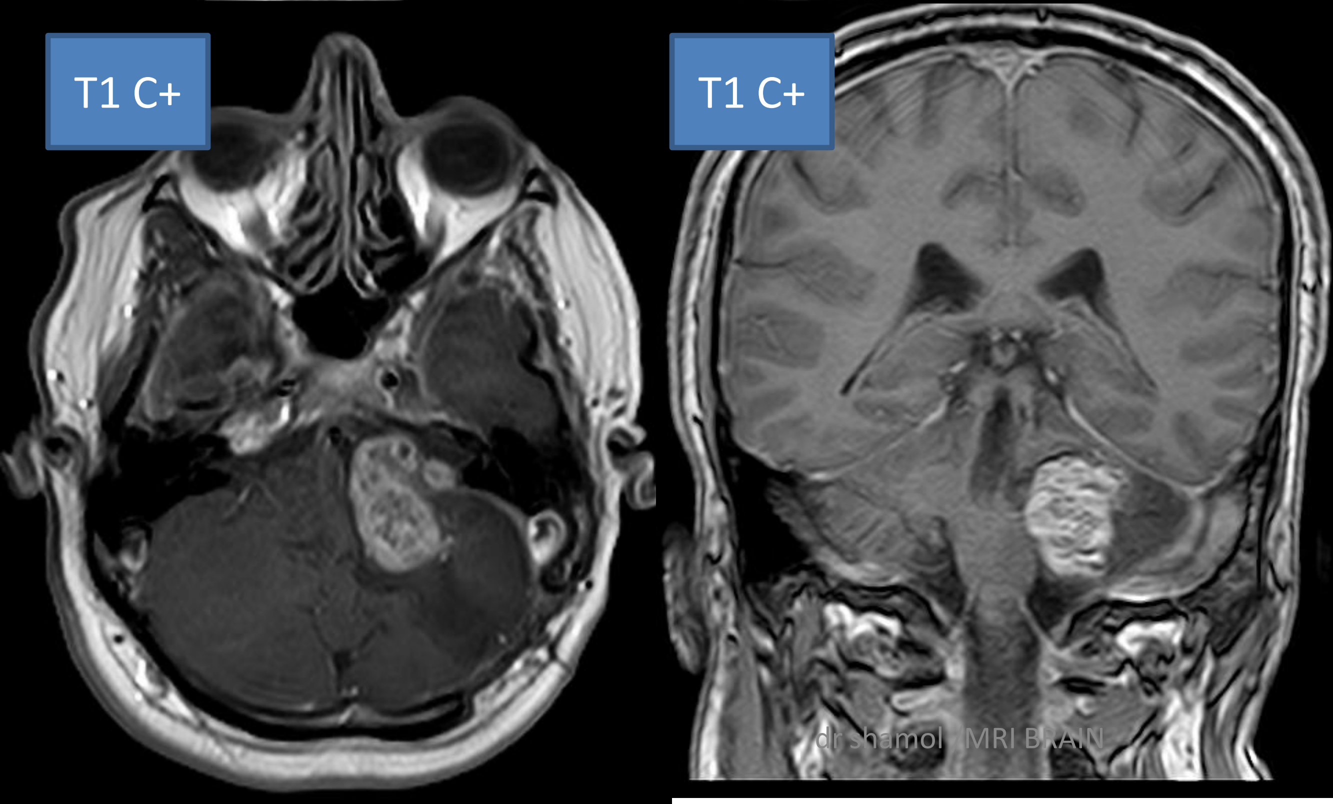


he mass is heterogeneosly hypointense on T1W, hyperintense on T2W and FLAIR sequences.

Acoustic schwannoma

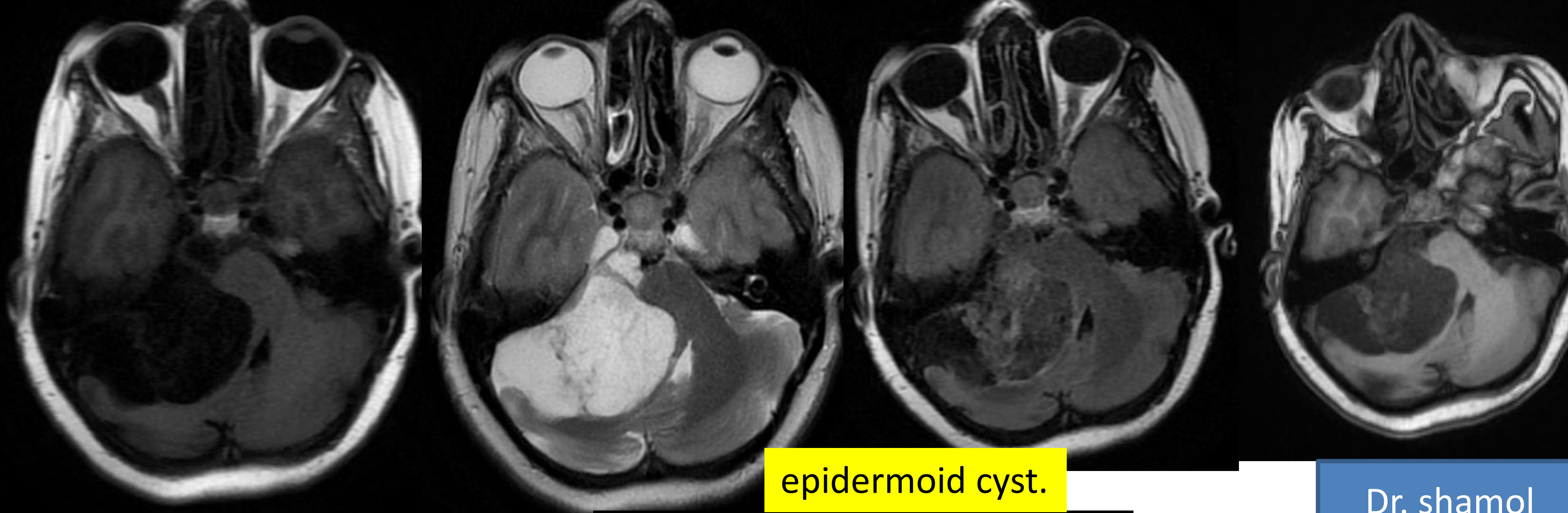
Dr.shamol





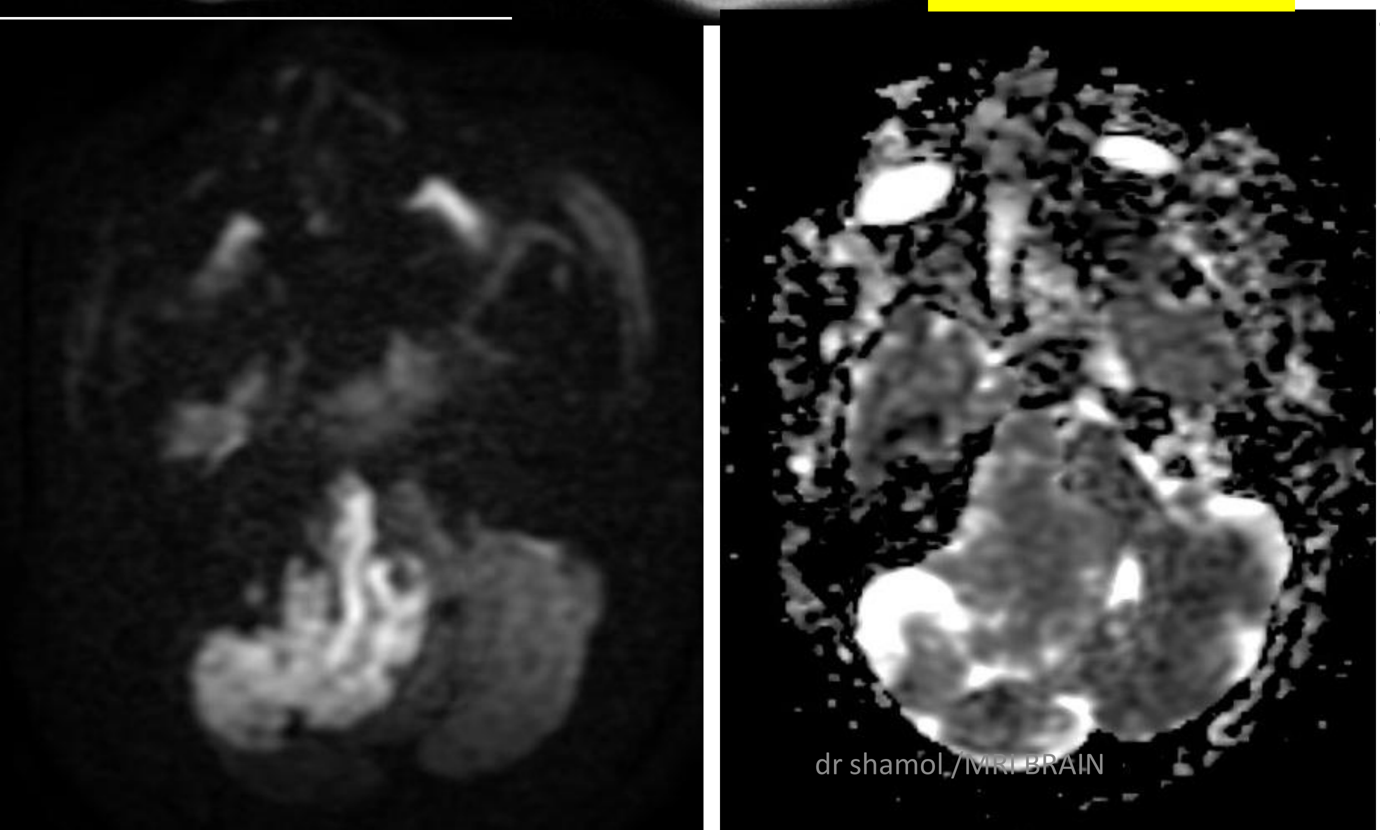
Vestibular schwannoma

Dr.shamol



epidermoid cyst.

Dr. shamol



T1

Hypointense

T1 C+ (Gd)

Post contrast scan shows no enhancement

T2

Hyperintense

FLAIR

often heterogeneous/dirty

DWI:

shows restricted diffusion and showing ADC similar to brain parenchyma.

Identify

pituitary gland
sella turcica

Determine Epicentre of the lesion

In, above, below or lateral to the sella

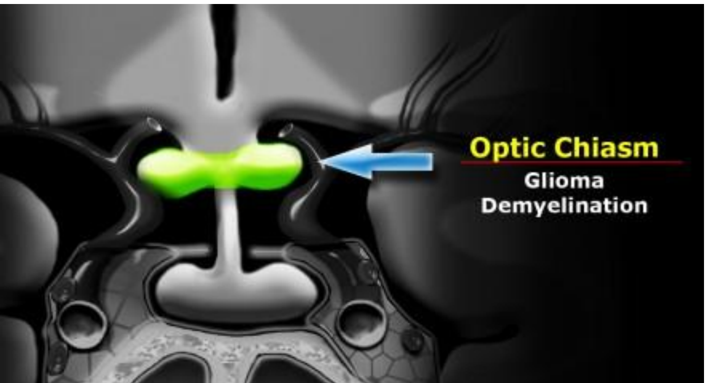
Analyse the lesion

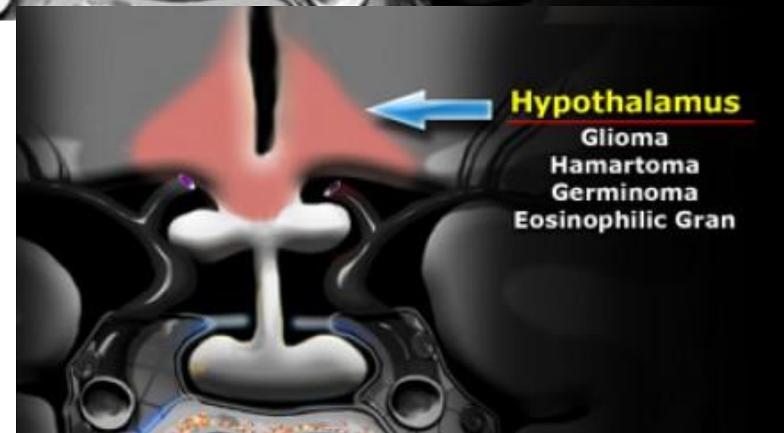
signal, cystic, solid
flow void, calcifications

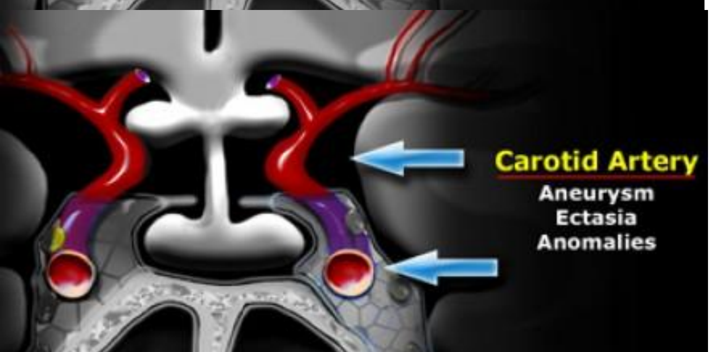
Differential Diagnosis

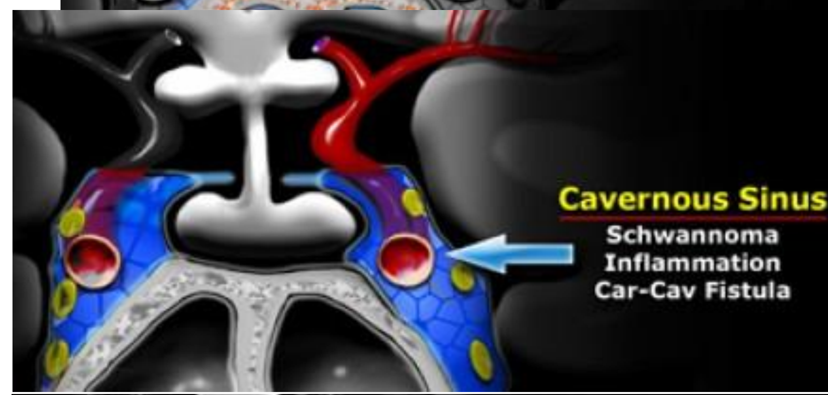

Pituitary Gland
Adenoma
Rathke Cleft Cyst
Craniopharyngioma

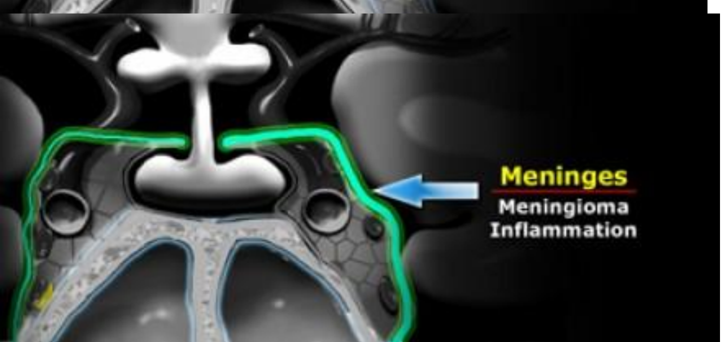

Pituitary Stalk
Rathke Cleft Cyst
Craniopharyngioma
Germinoma
Eosinophilic Gran.
Metastasis

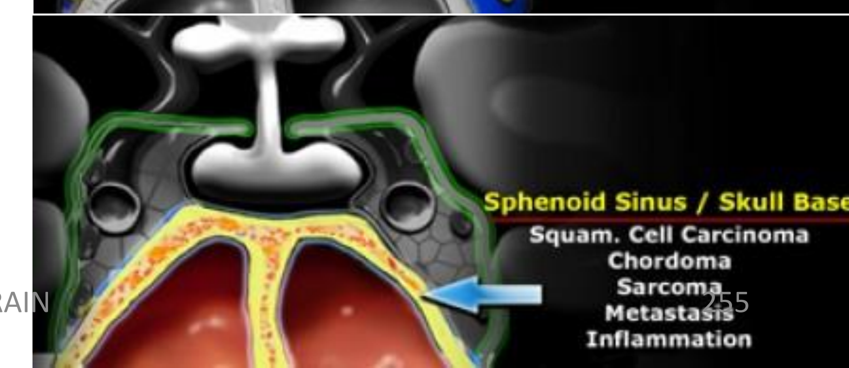

Optic Chiasm
Glioma
Demyelination

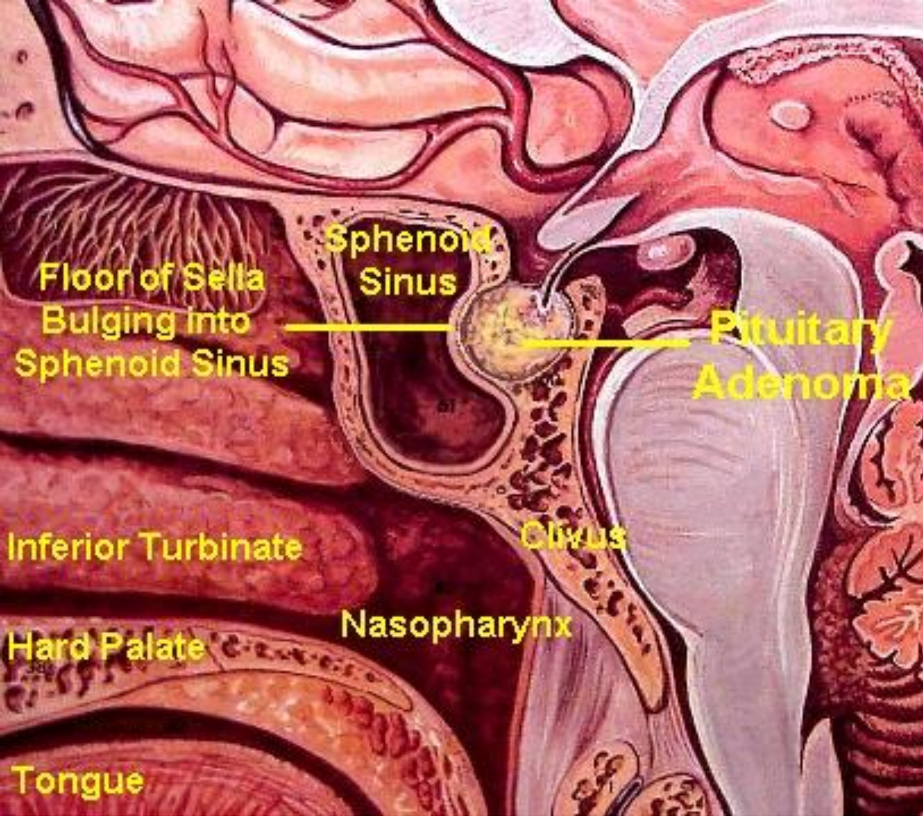

Hypothalamus
Glioma
Hamartoma
Germinoma
Eosinophilic Gran

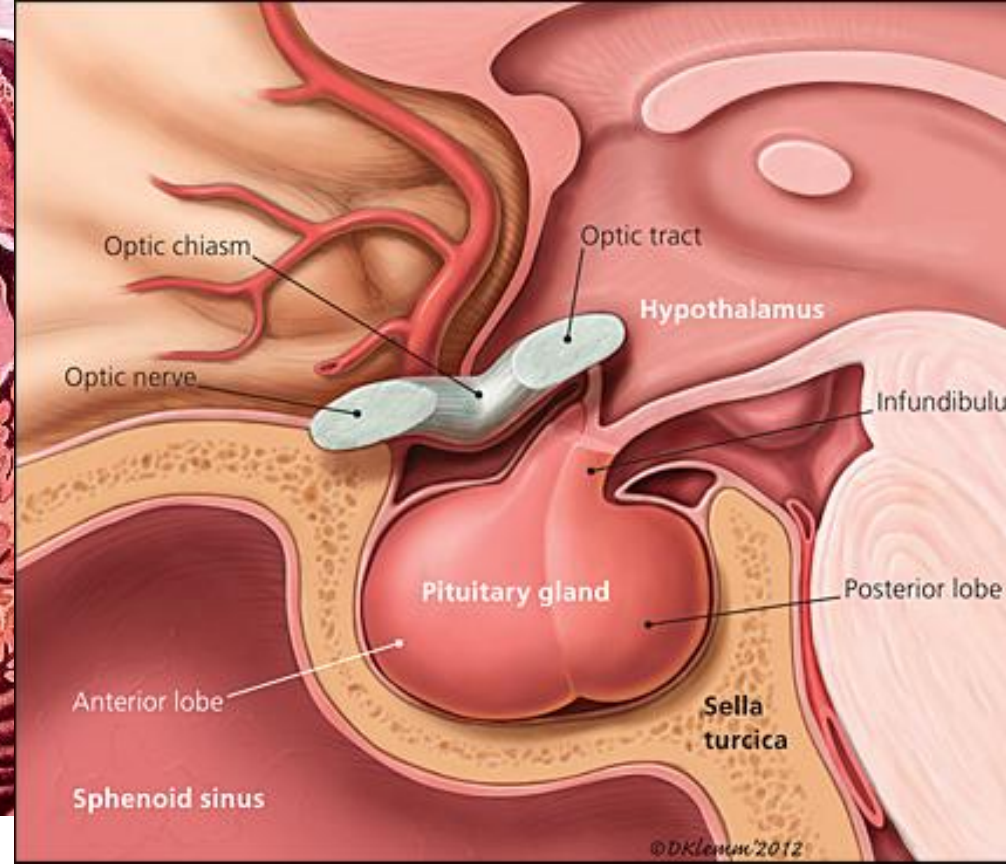

Carotid Artery
Aneurysm
Ectasia
Anomalies


Cavernous Sinus
Schwannoma
Inflammation
Car-Cav Fistula


Meninges
Meningioma
Inflammation


Sphenoid Sinus / Skull Base
Squam. Cell Carcinoma
Chordoma
Sarcoma
Metastasis
Inflammation





PITUITARY ADENOMA

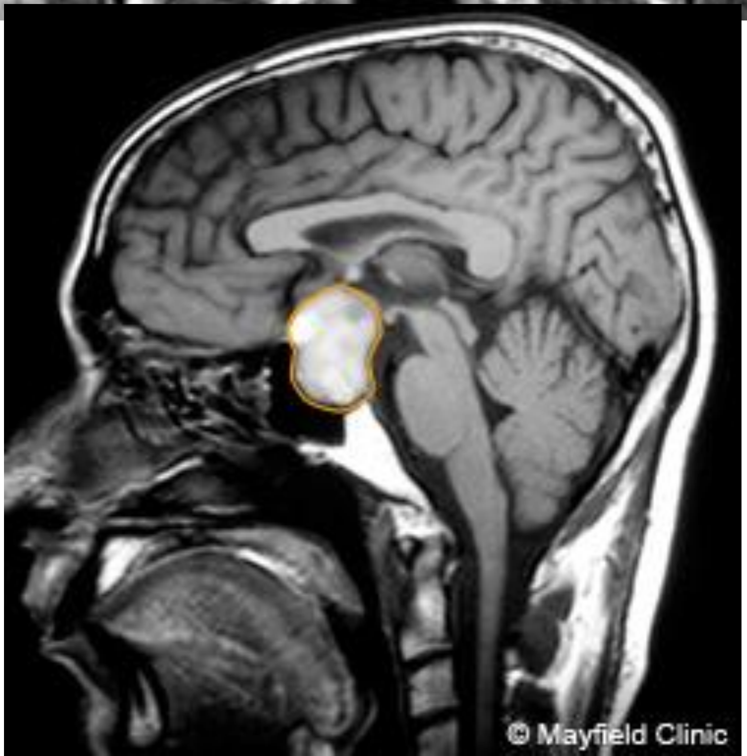
Classification

Microadenoma	< 10 mm diameter	Secreting adenoma
Macroadenoma	> 10 mm diameter	Mass effects Non secreting

Pituitary macroadenoma

T1	**typically isointense to grey matter**
	larger lesions are often heterogeneous and vary in signal due to areas of cystic change/necrosis/haemorrhage
T1 C+ (Gd)	solid components demonstrate moderate to bright enhancement
T2	typically isointense to grey matter
	larger lesions are often heterogeneous and vary in signal due to areas of cystic change/necrosis/haemorrhage


Tumor invading beyond cavernous sinus
Optic chiasm
Pituitary gland
Carotid arteries (red) in normal cavernous sinus (blue)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Organ:HEAD
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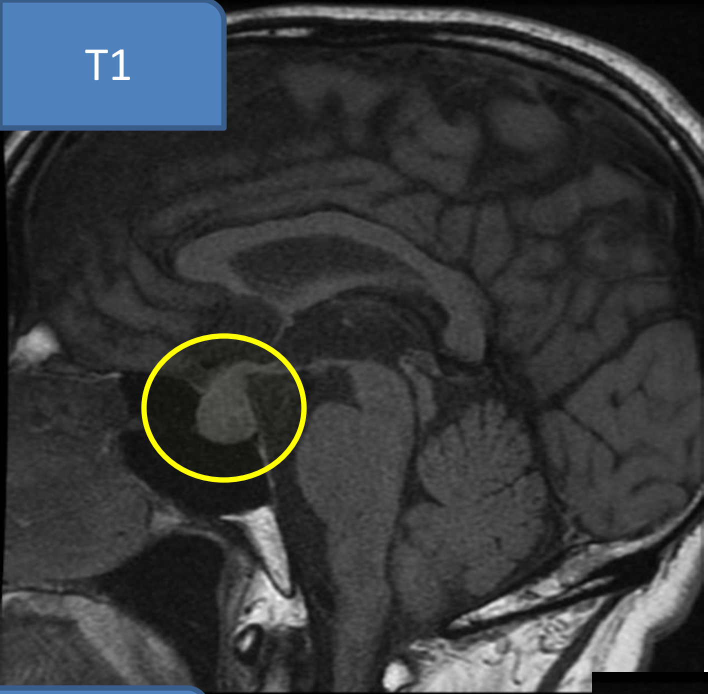
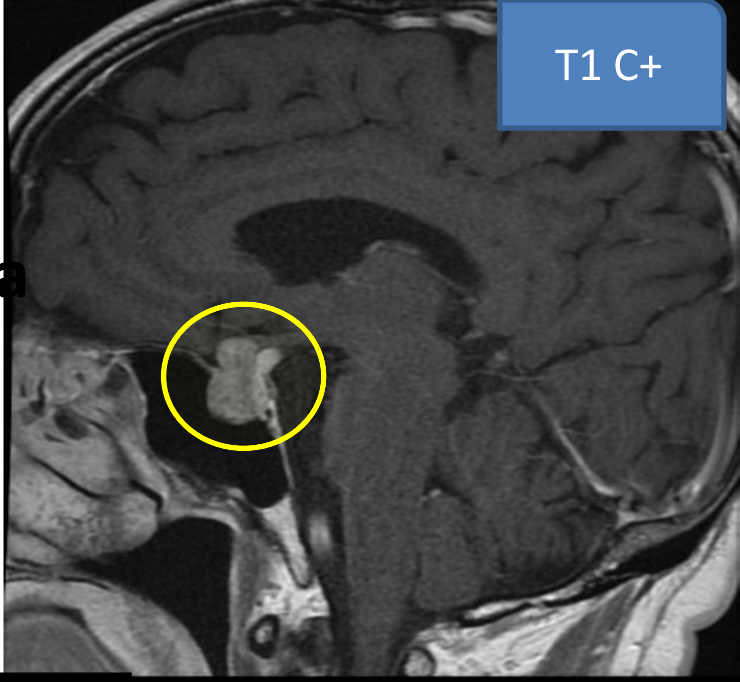
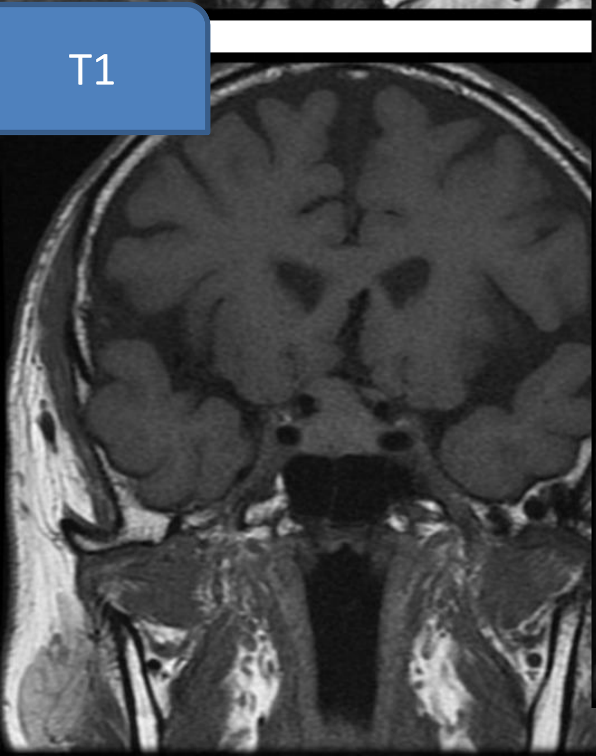
Dr.shamol

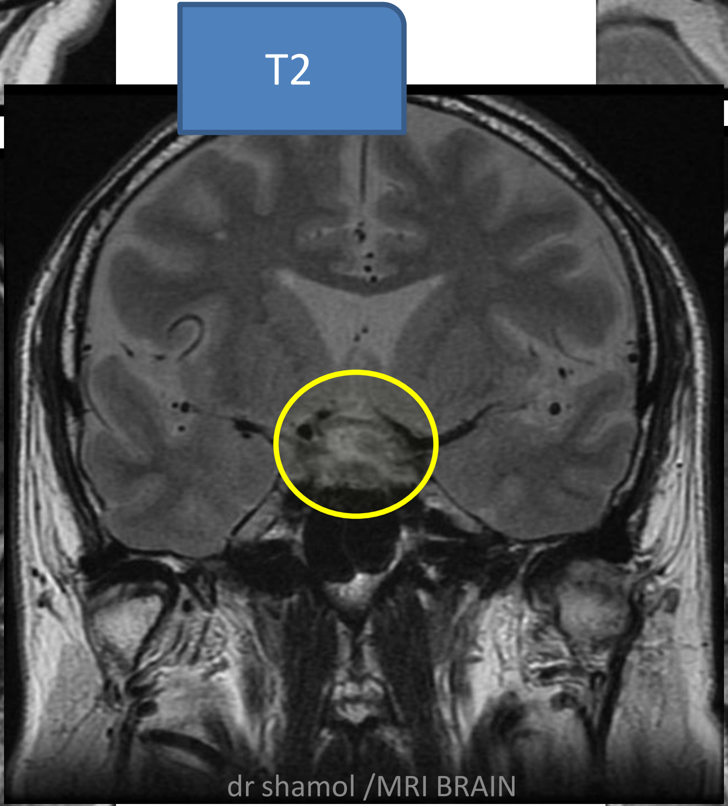
CASE 1

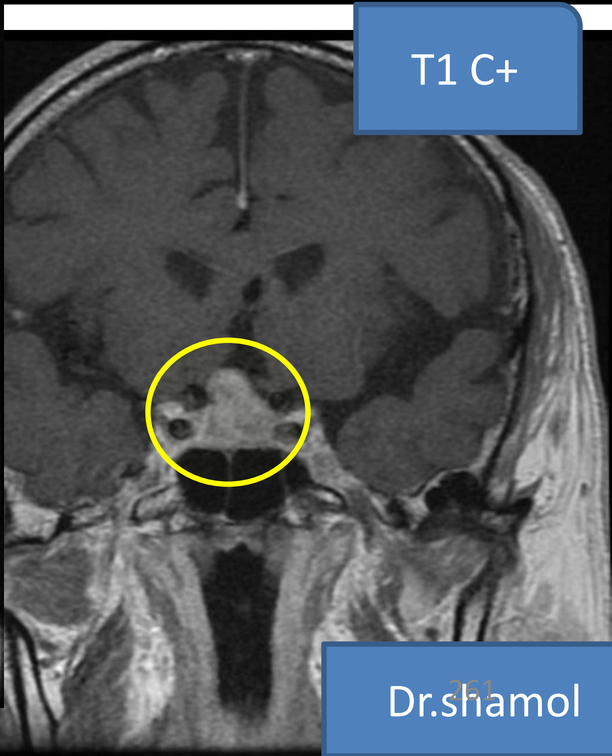
Pituitary macroadenoma

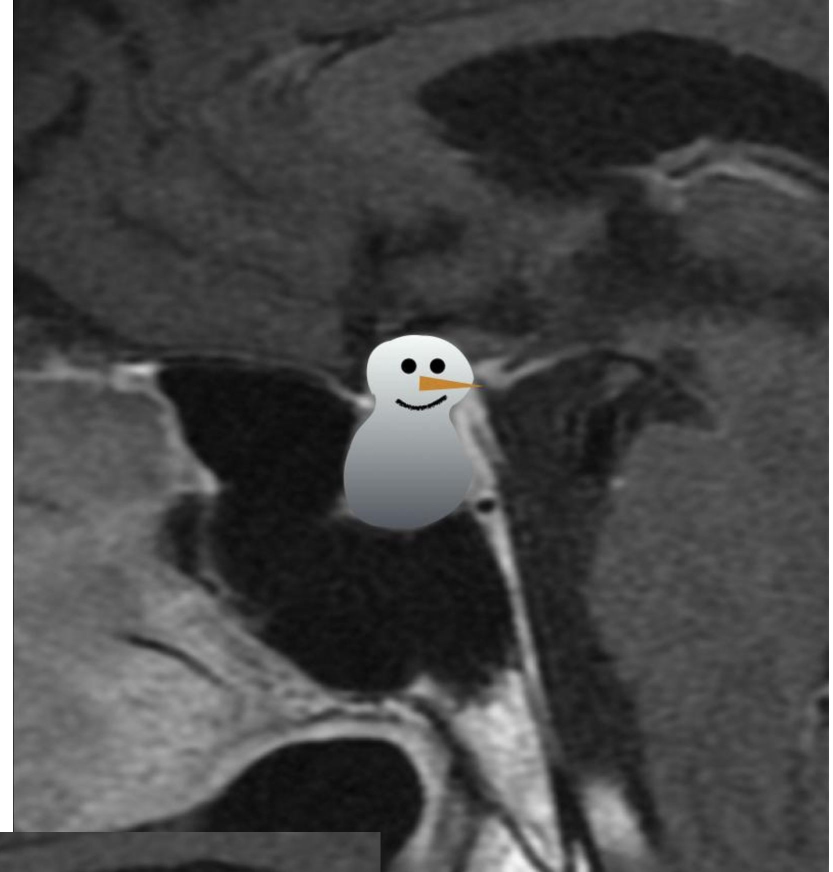


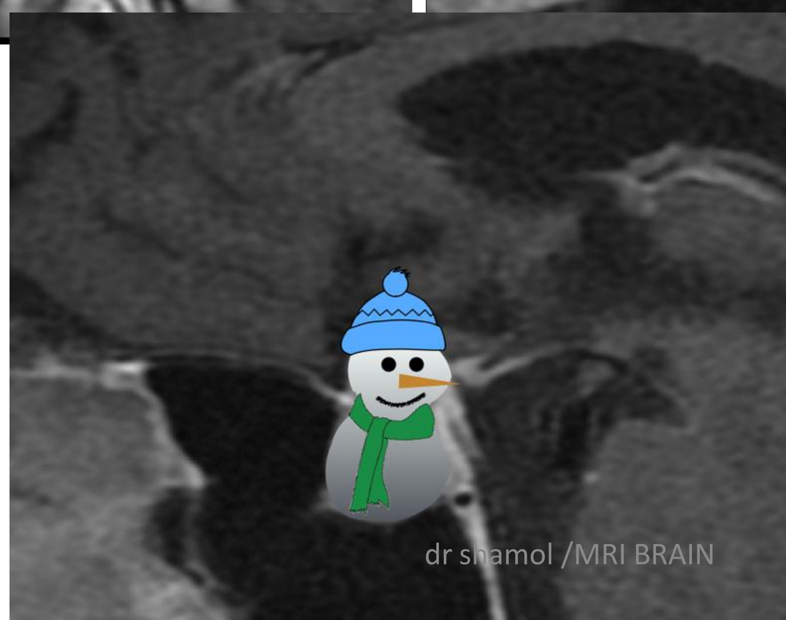






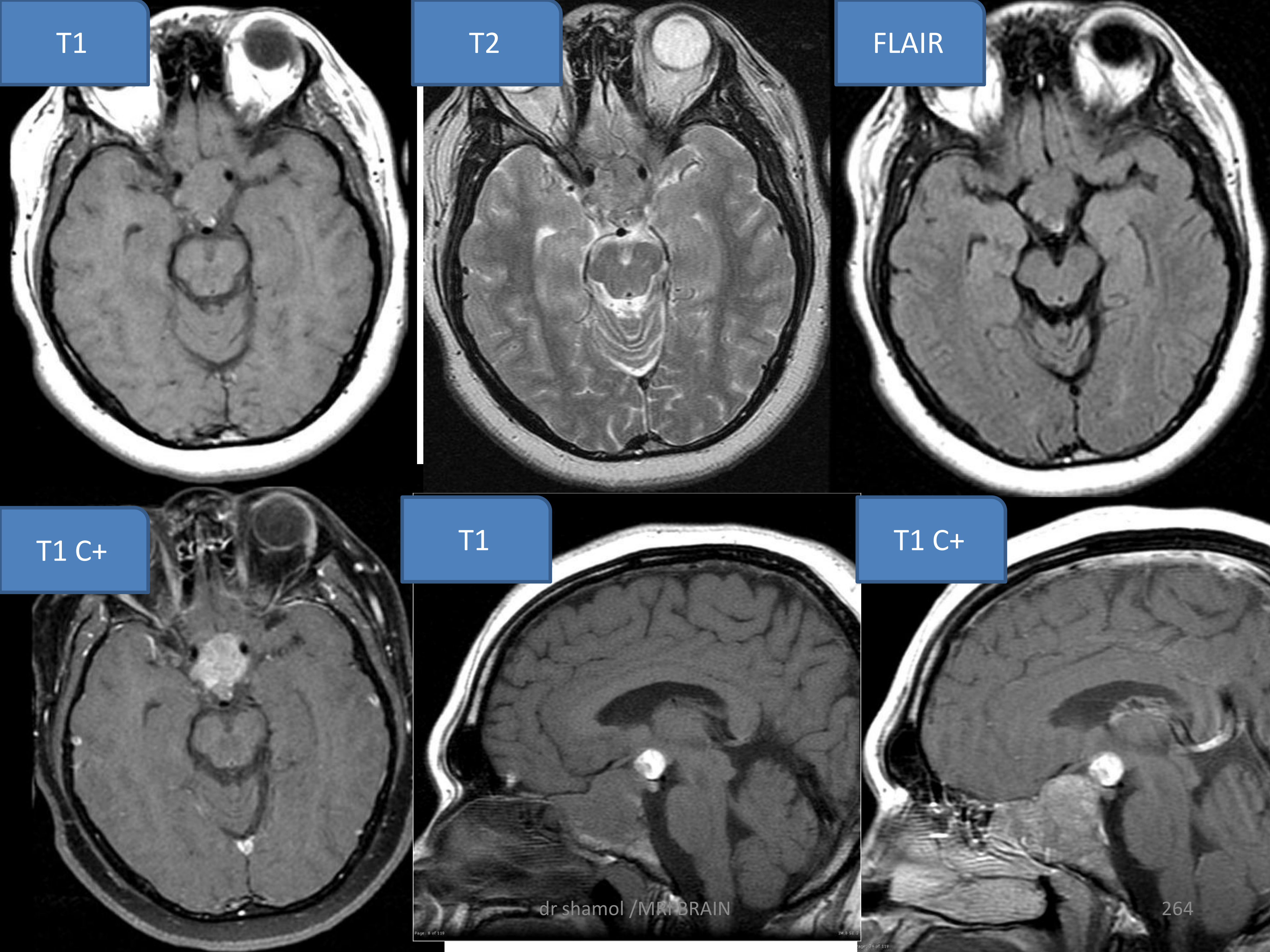


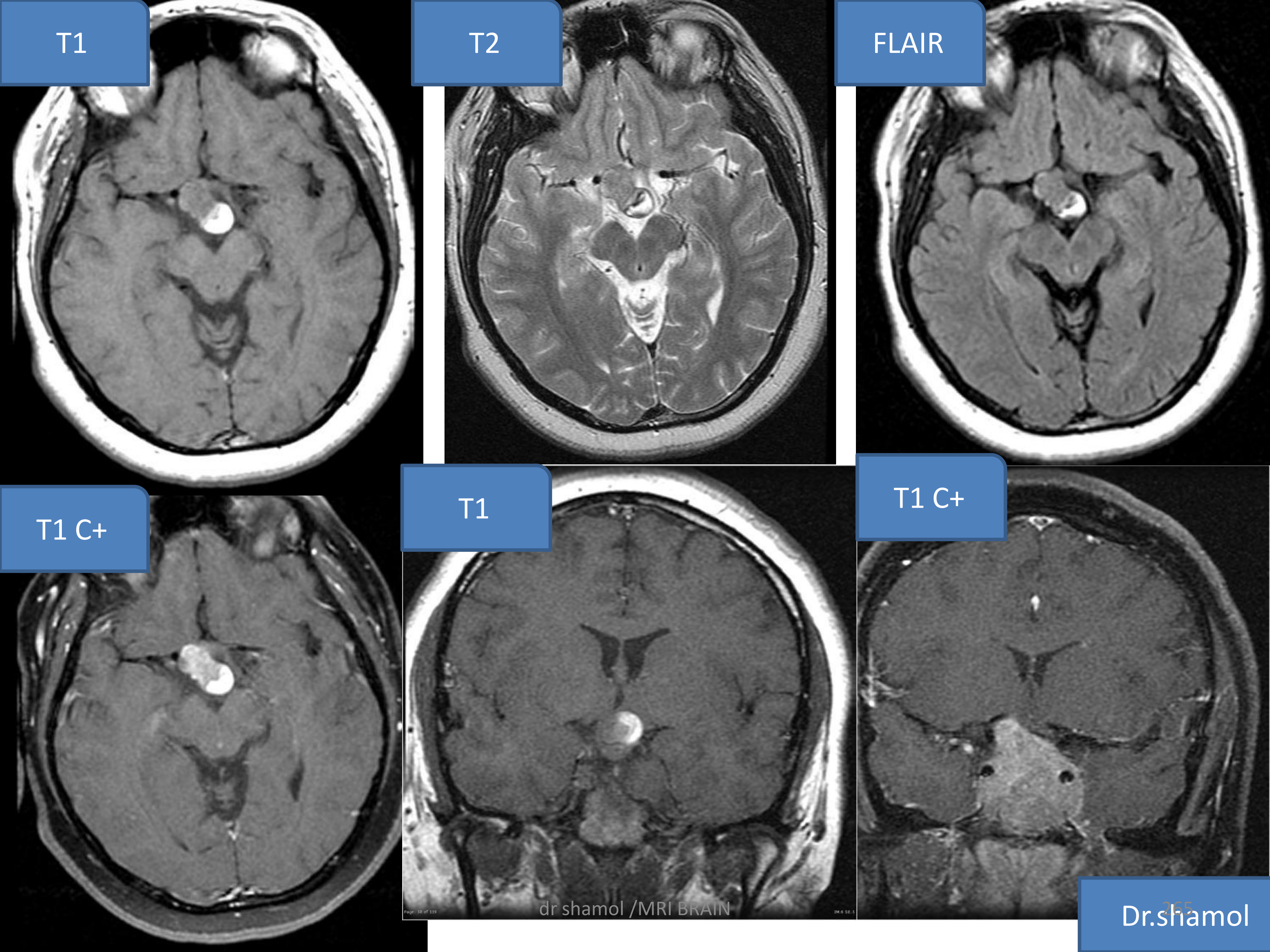




Annotated image demonstrating the indentation at the diaphragma sellae that gives a snowman or figure eight configuration to the macroadenoma.

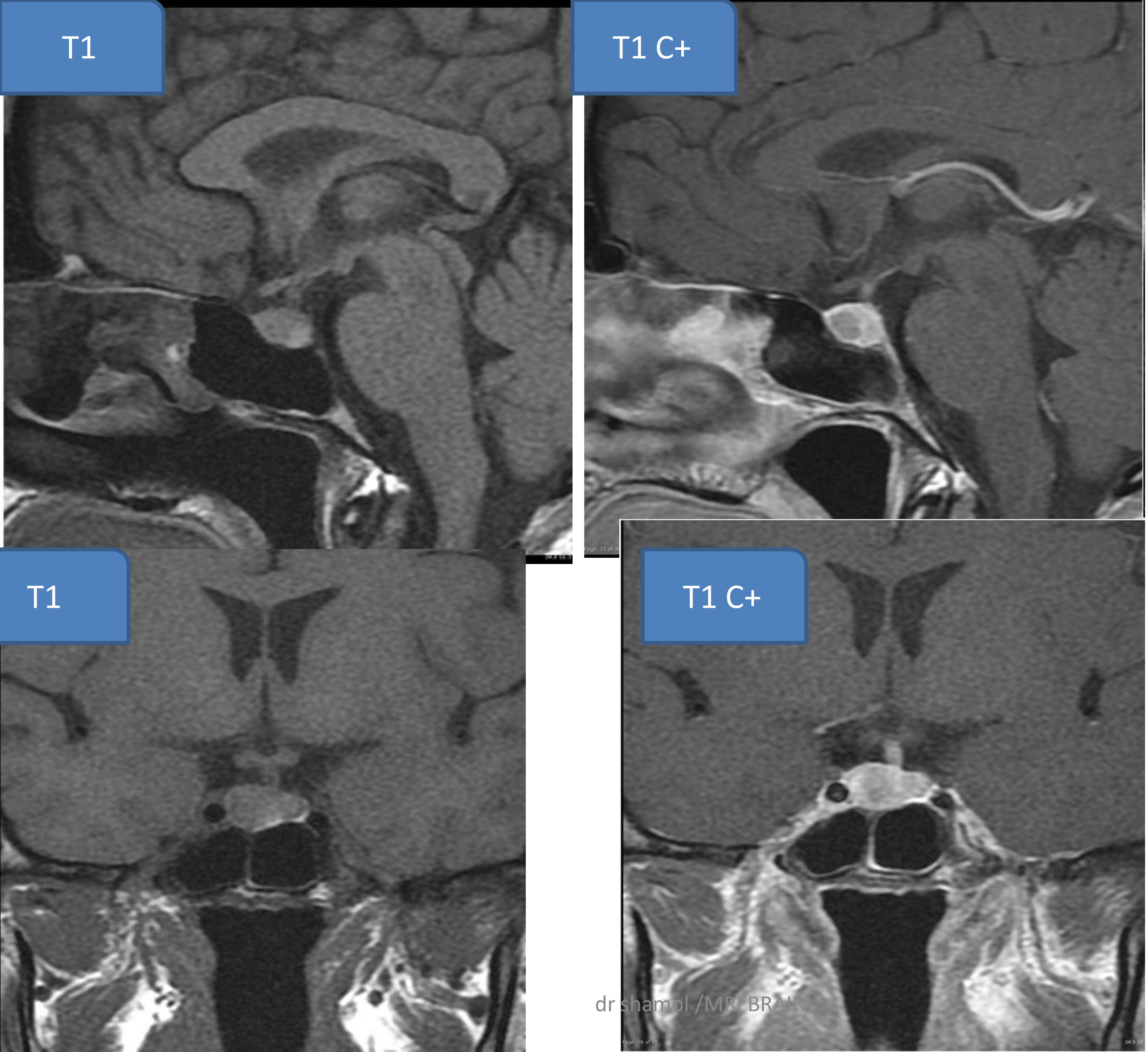
CASE 1

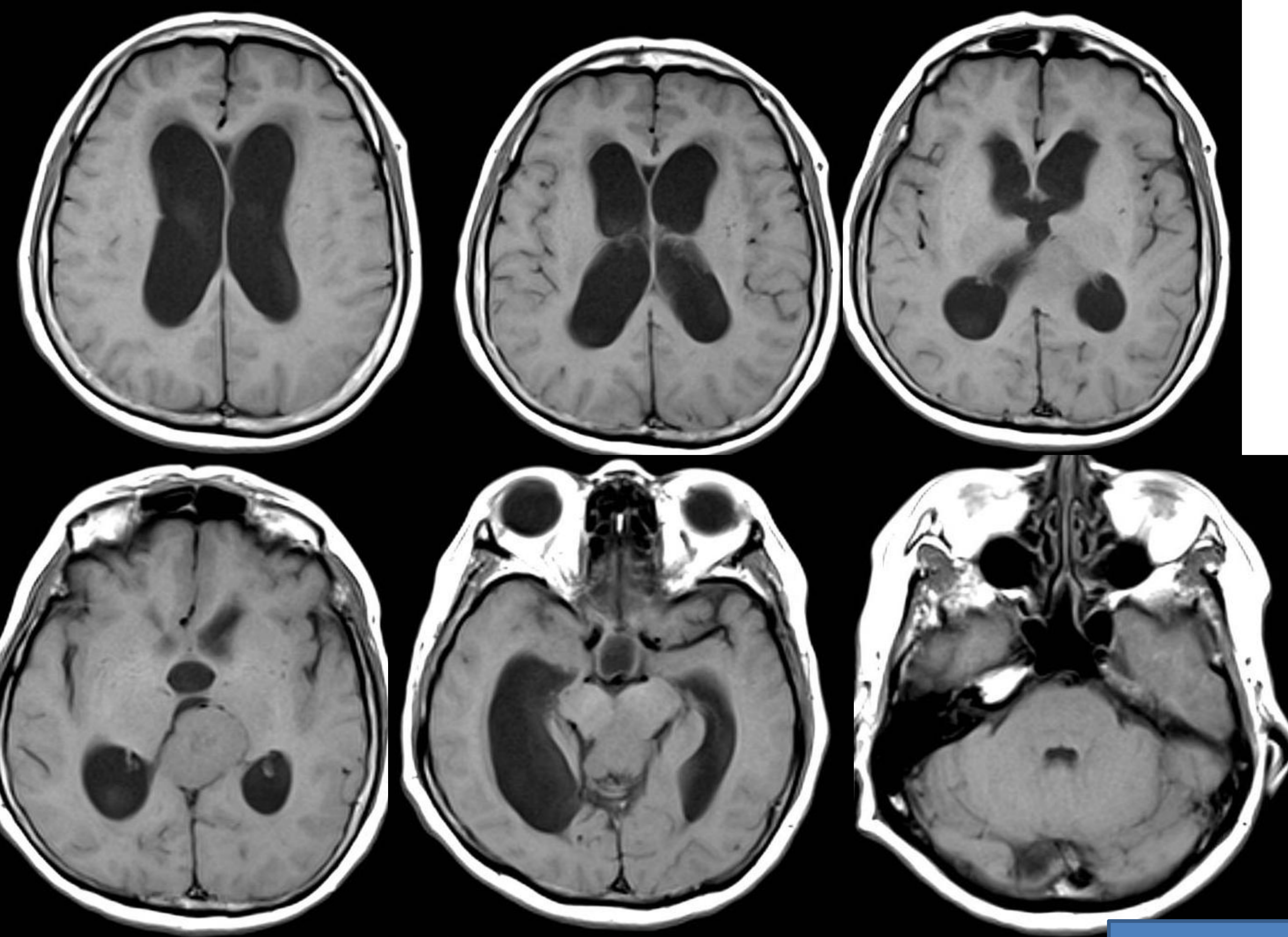

T1
T2
FLAIR
T1 C+
T1
T1 C+

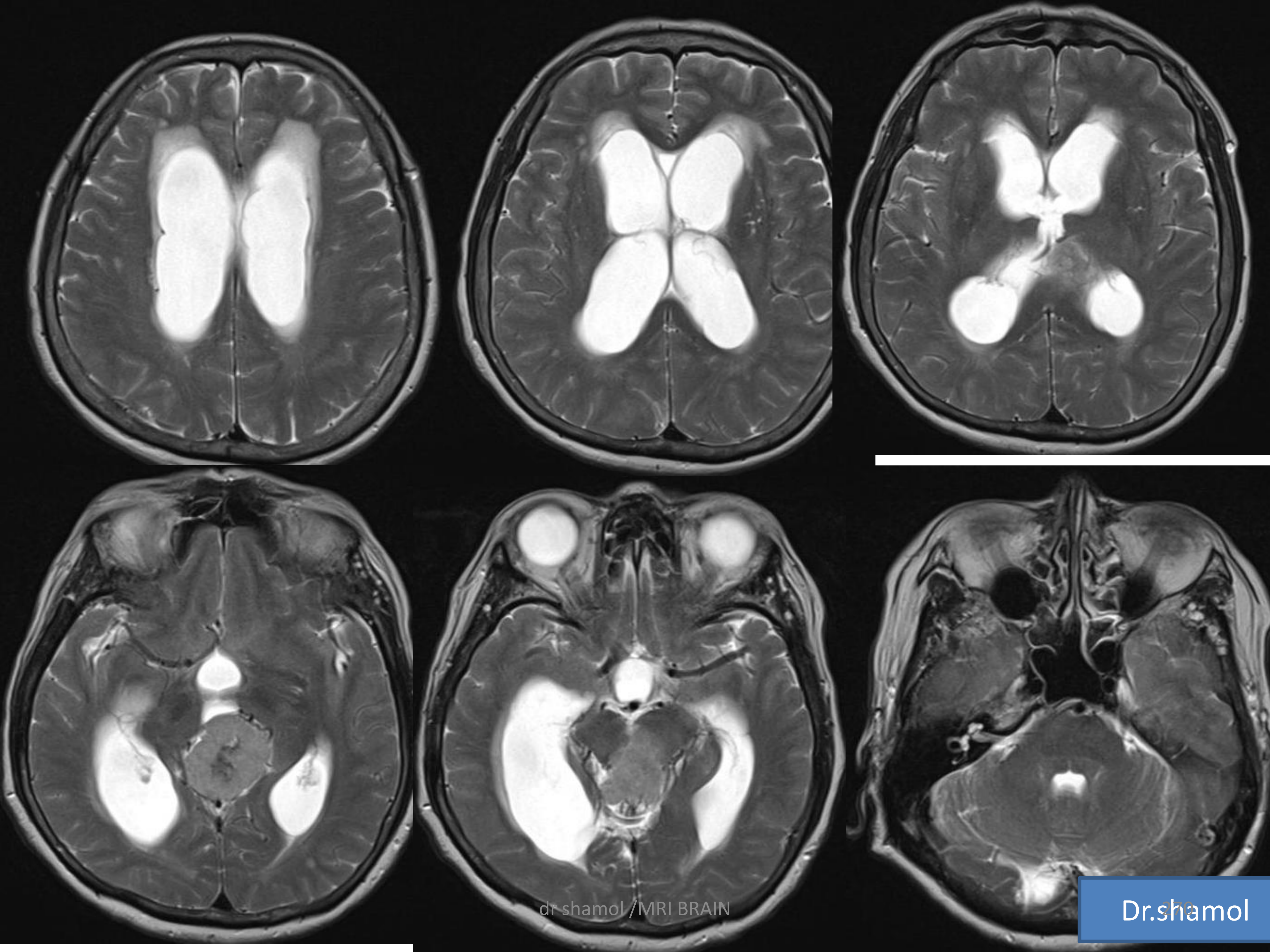

T1
T2
FLAIR
T1 C+
T1
T1 C+
Dr.shamol

Pituitary microadenoma

T1	usually isointense to normal pituitary
T1 C+ (Gd)	dynamic sequences demonstrate a rounded region of delayed enhancement compared to the rest of the gland [1]
	delayed images are variable, ranging from hypo-enhancement (most common) to isointense to the rest of the gland, to hyperintense (retained contrast)
T2	variable, but often a little hyperintense

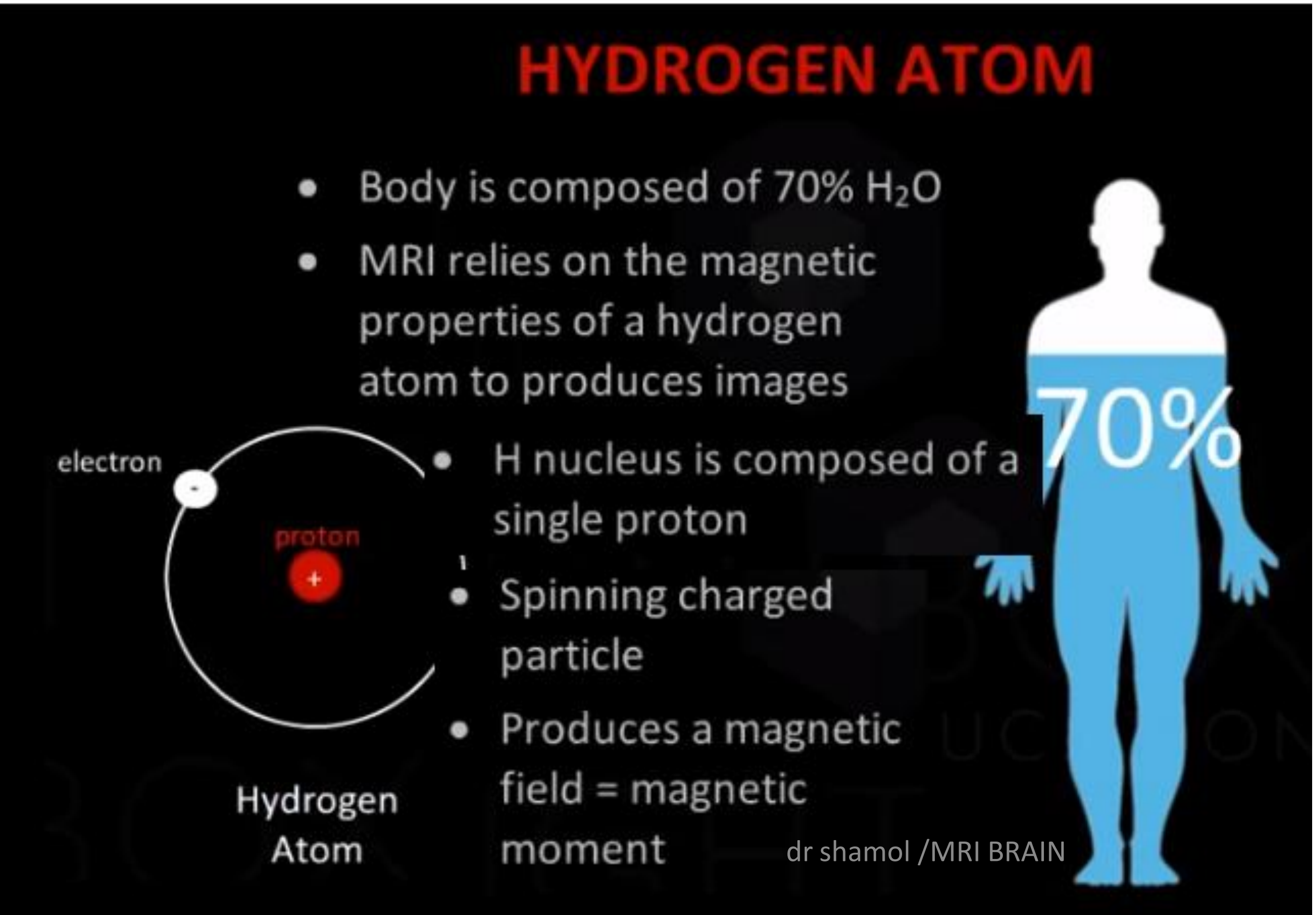

T1
T1 C+
T1
T1 C+





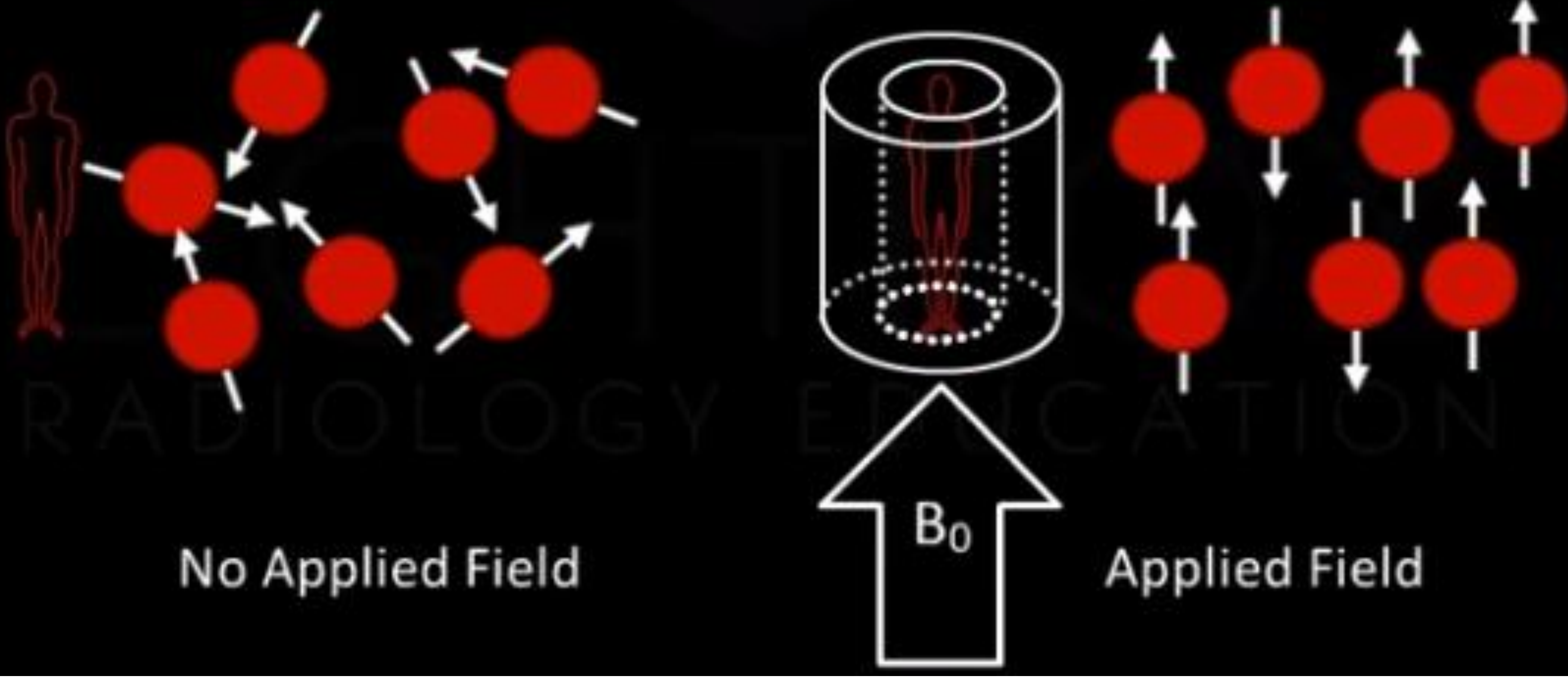
Basic of mri

- ❖Hydrogen is the simplest element with atomic number of 1 and atomic weight of 1
- ❖When in ionic state (H+), it is nothing but a proton.
- ❖Proton is not only positively charged, but also has magnetic spin (wobble)
- ❖We are magnets but we can’t act like magnets?
- ❖Because The protons (i.e. Hydrogen ions) in body are spinning in a hapazard fashion, and cancel all the magnetism. That is our natural state
- ❖MRI utilizes this magnetic spin property of protons of hydrogen to elicit images



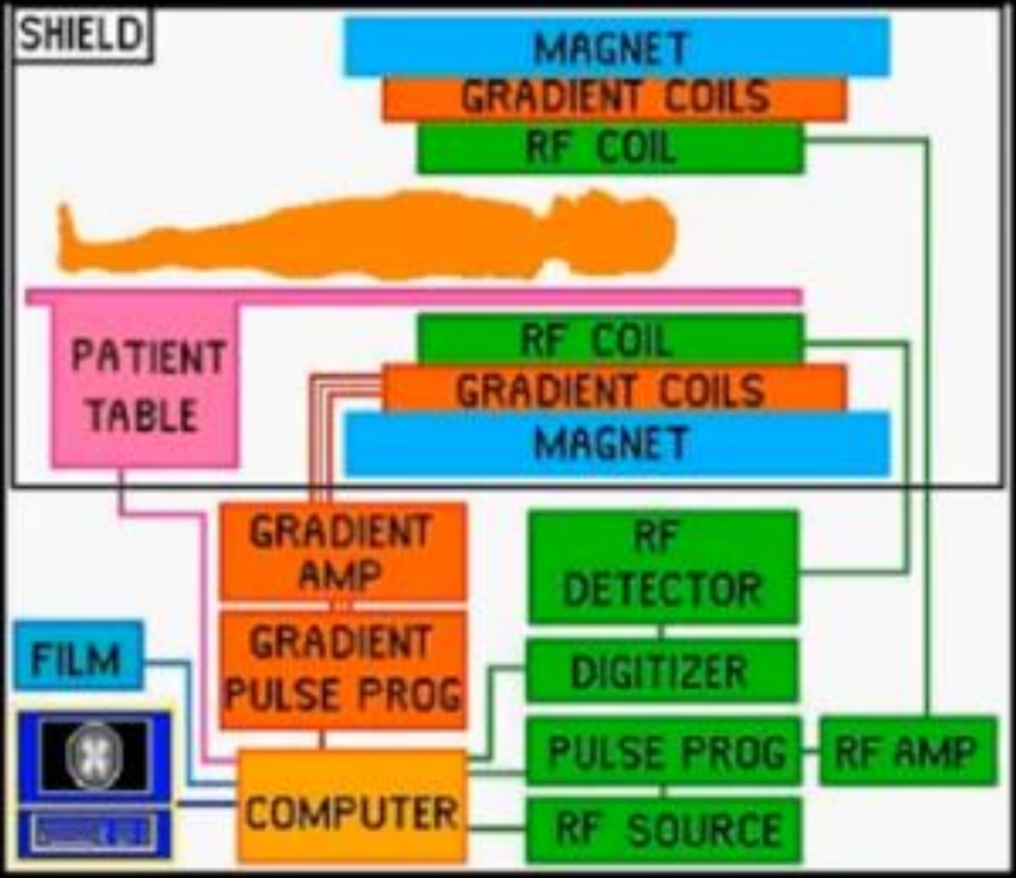
PRIMARY MAGNETIC FIELD

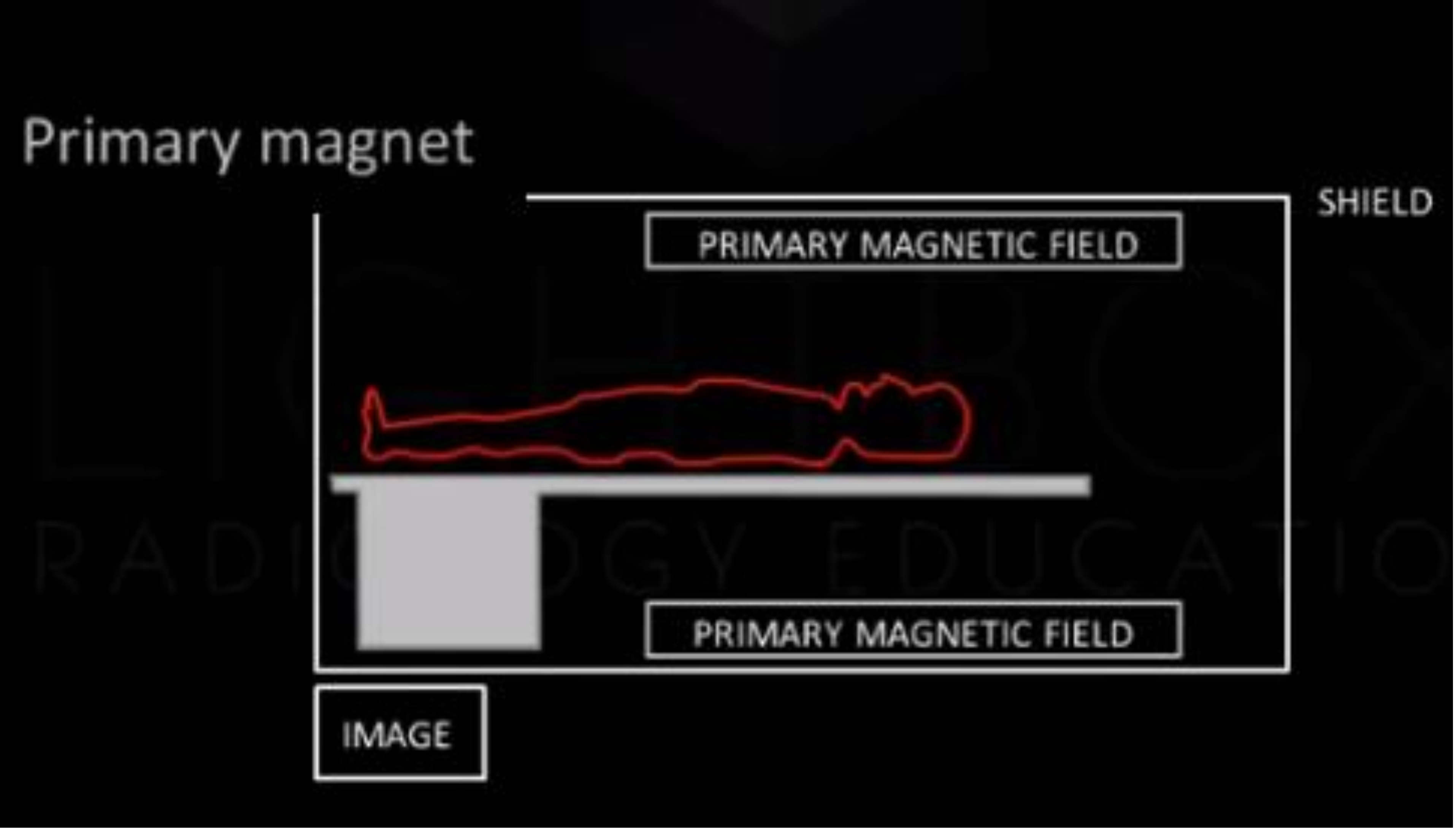
- Superconducting magnet
- Constant 1.5 - 3T
- Hydrogen atoms align parallel or antiparallel to the primary field (Bo) = longitudinal magnetisation



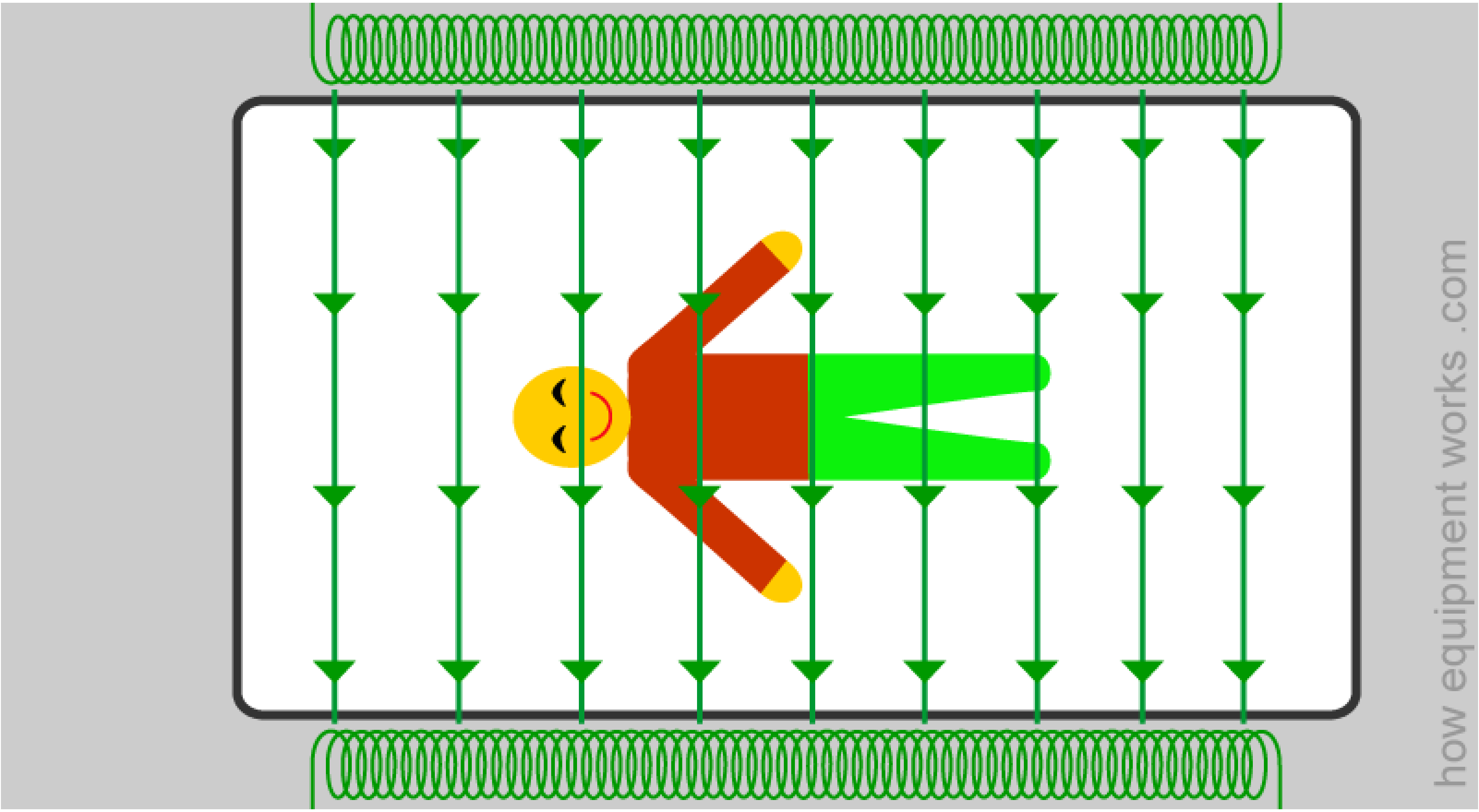
MRI Hardware

- Magnet 1.5T or 3.0T
- Radio Frequency Coil
- Gradient Coils
- Computer

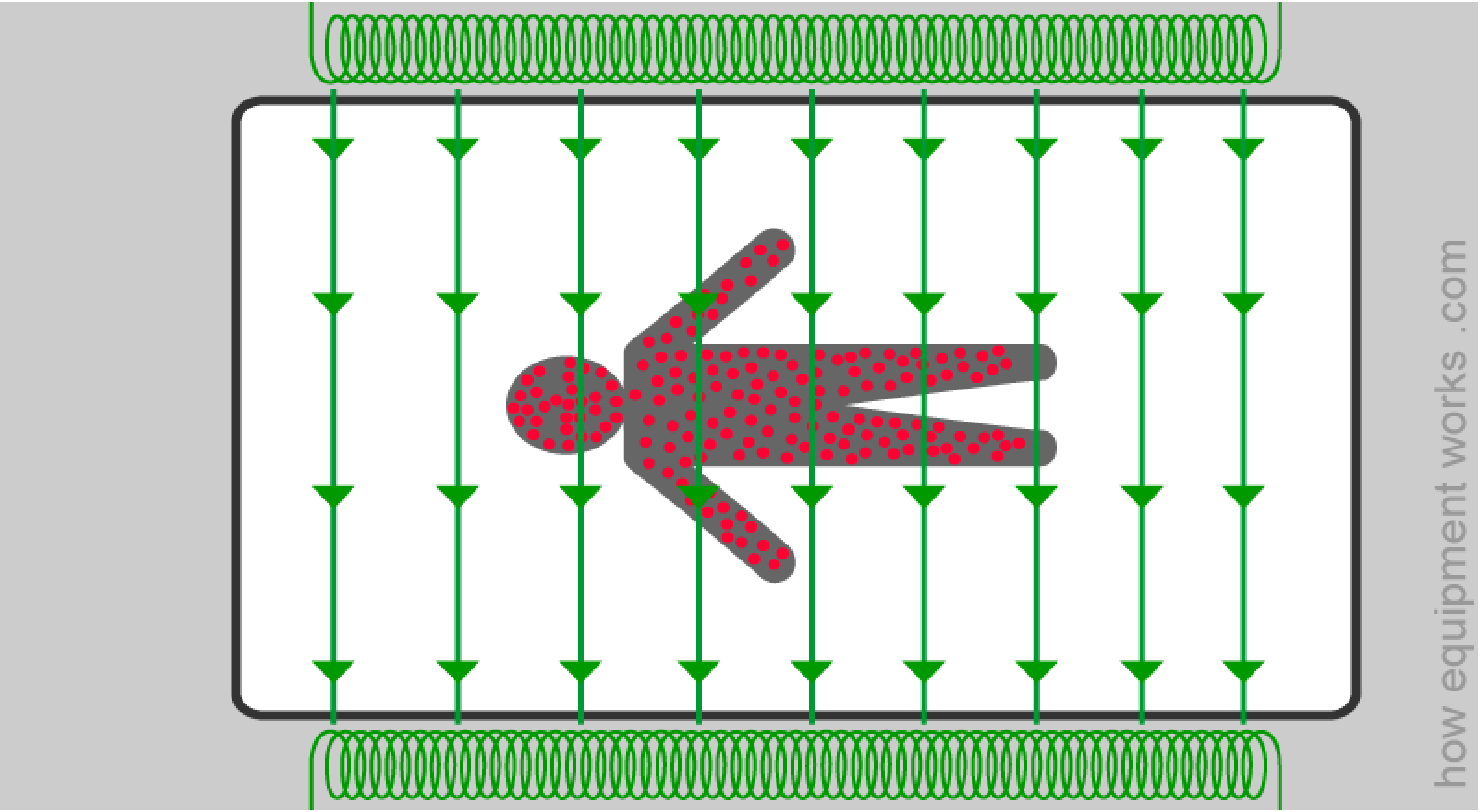



Primary magnet
SHIELD
PRIMARY MAGNETIC FIELD
PRIMARY MAGNETIC FIELD
IMAGE

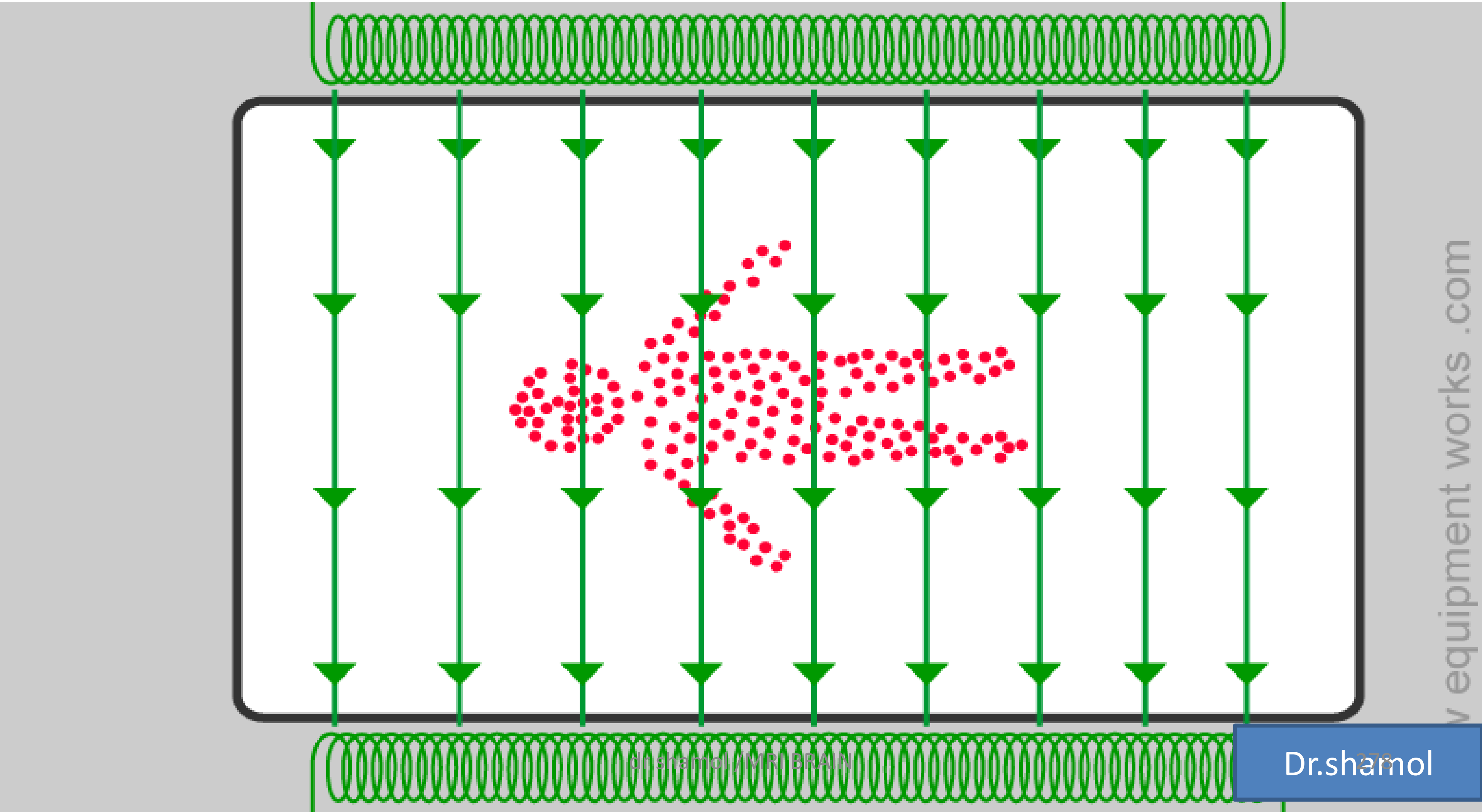
We now put the patient into the magnetic field of the MRI machine.



The patient, like all of us, has water molecules distributed all over. As described before, the water molecules have hydrogen nuclei, and this is what is of interest to the MRI machine.



To make the diagrams clearer . Instead Of patient,we will show the hydrogen nuclei that are inside him



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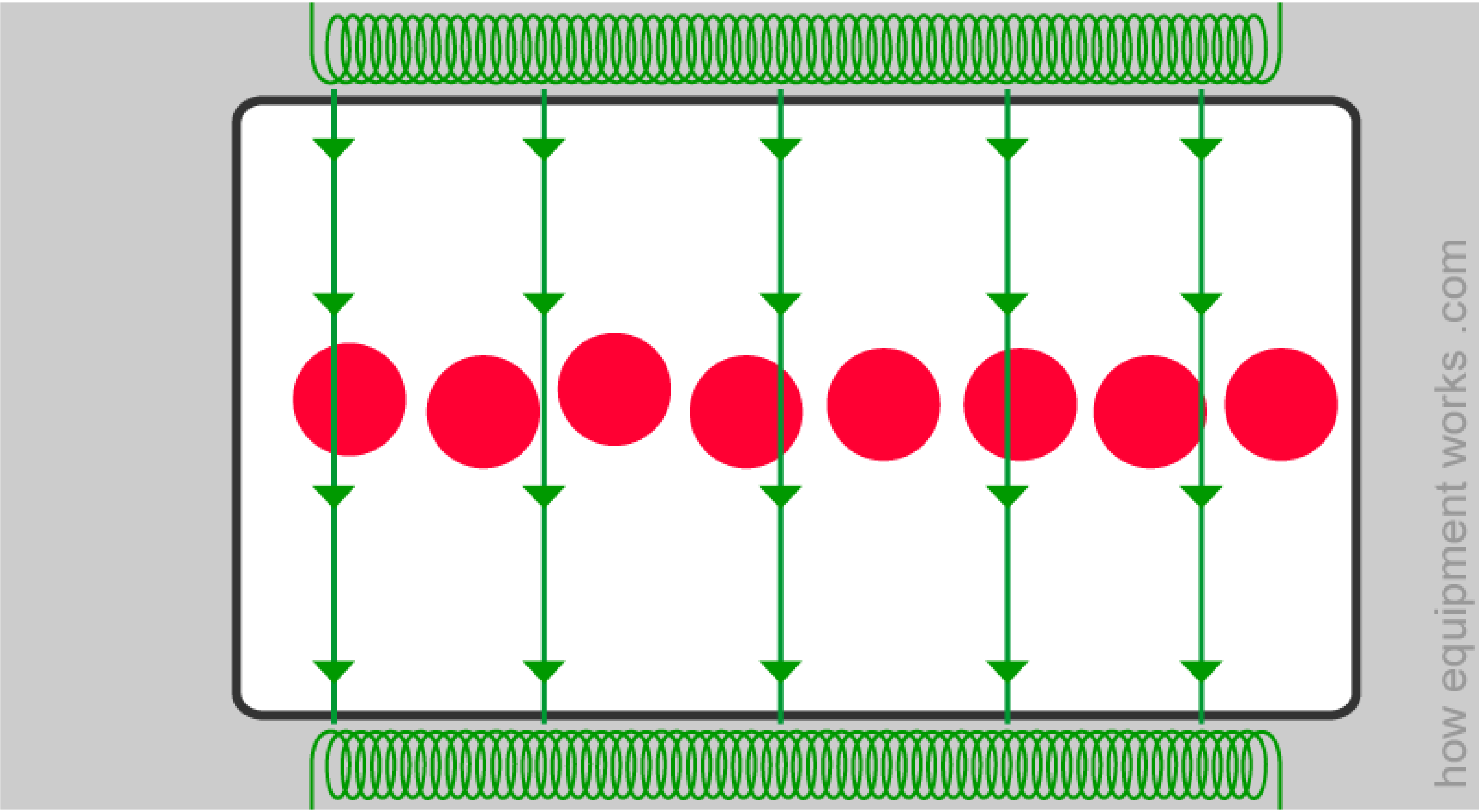
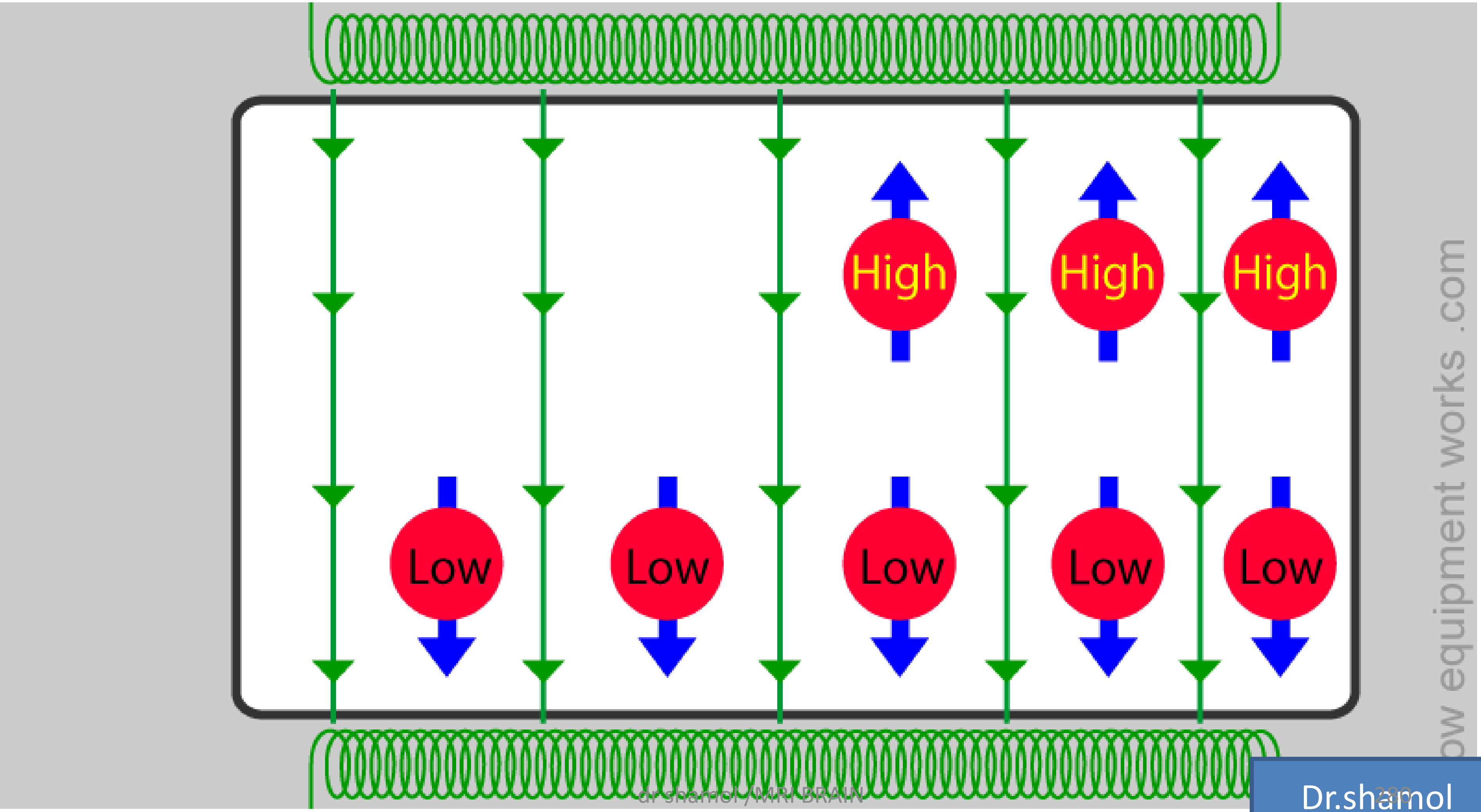
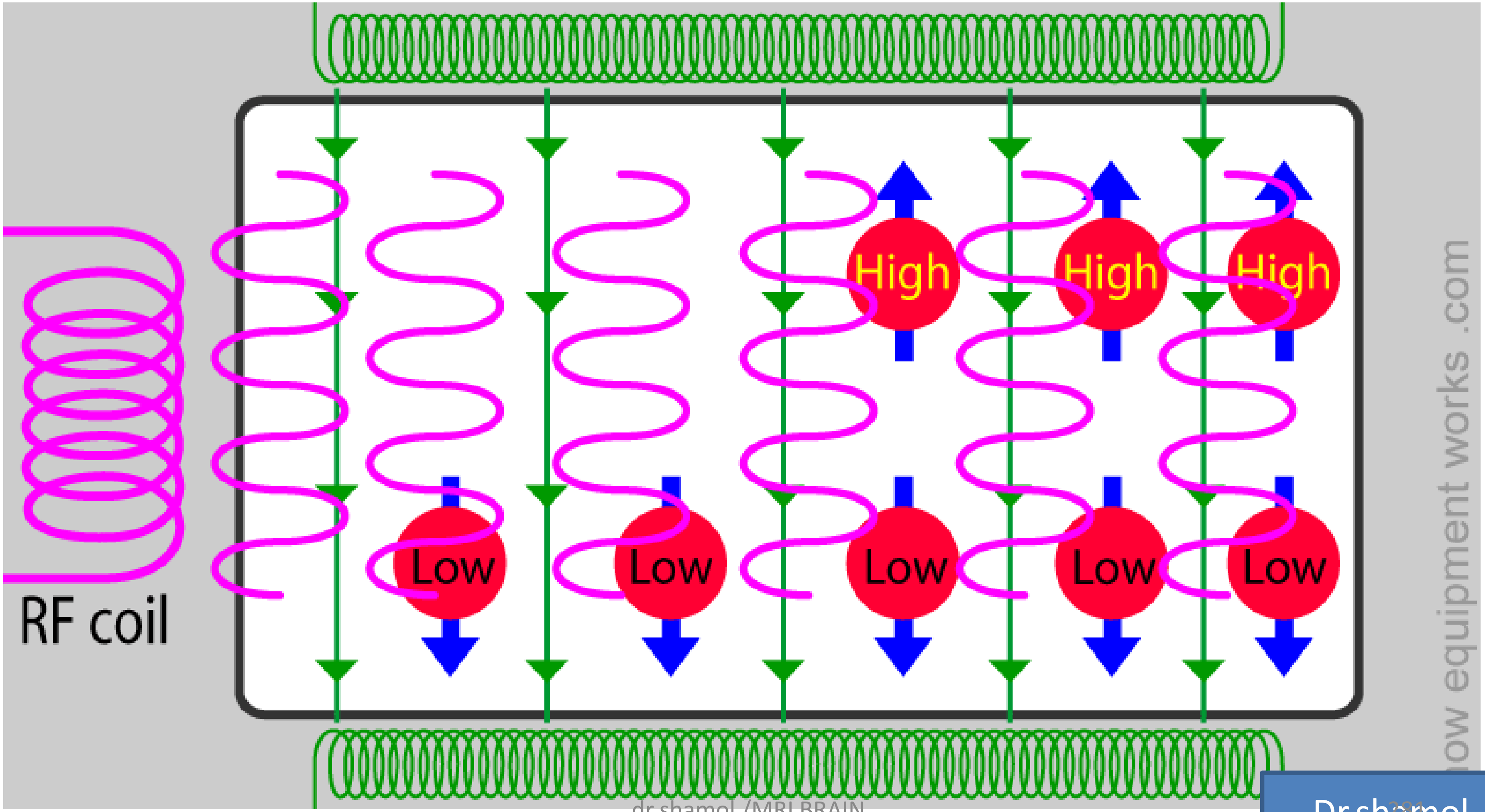
To further make the diagrams clearerwe will showing a close up of only a few of the hydrogen nuclei



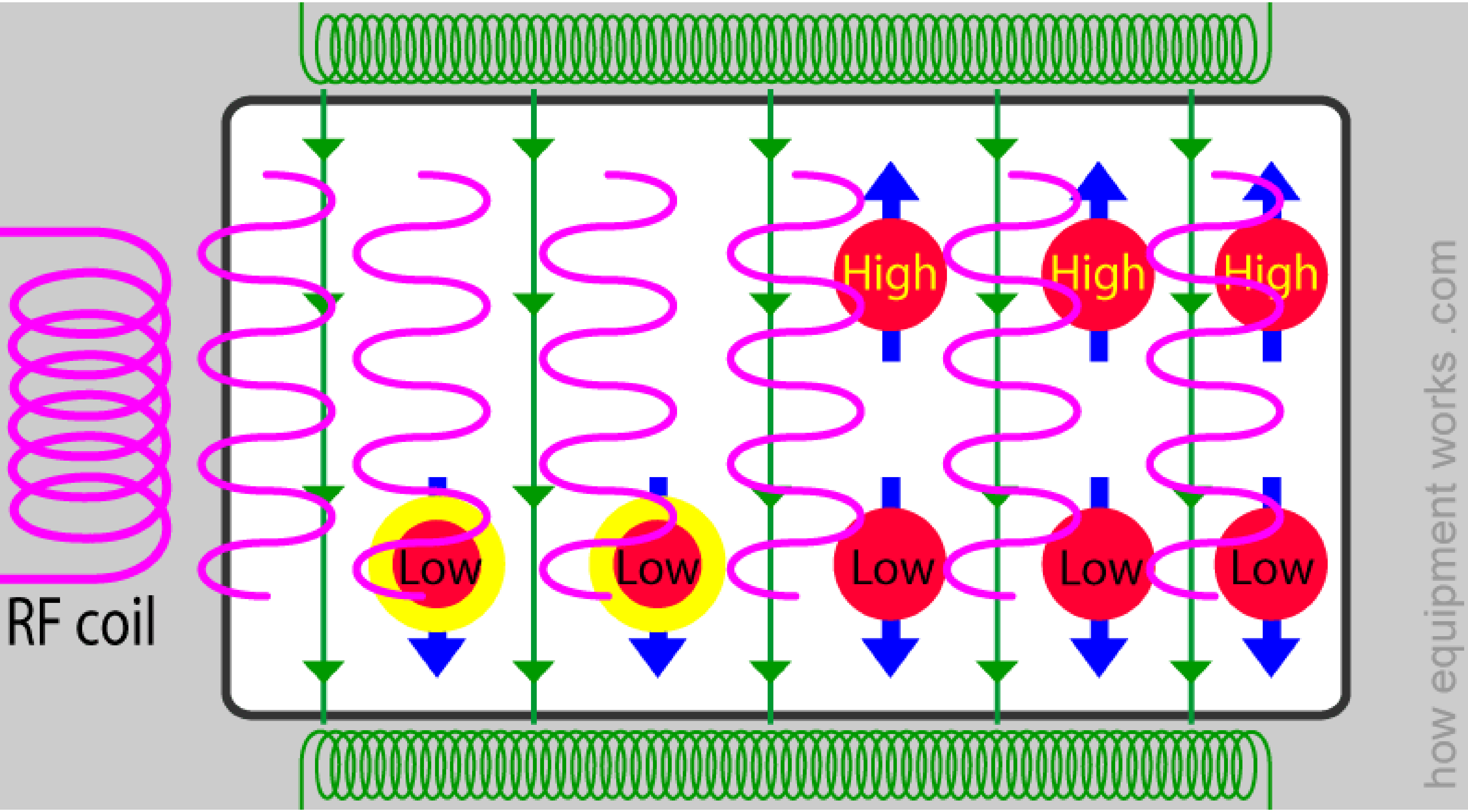
the magnetic field (green lines) are going from the top to the bottom. The strong magnetic field makes the spins (blue arrows) of the hydrogen nuclei line up along the magnetic field. Some of the hydrogen nuclei line up in the direction of the magnetic field (lower nuclei in diagram) and other hydrogen nuclei line up opposite to the direction of the magnetic field (upper nuclei in diagram).



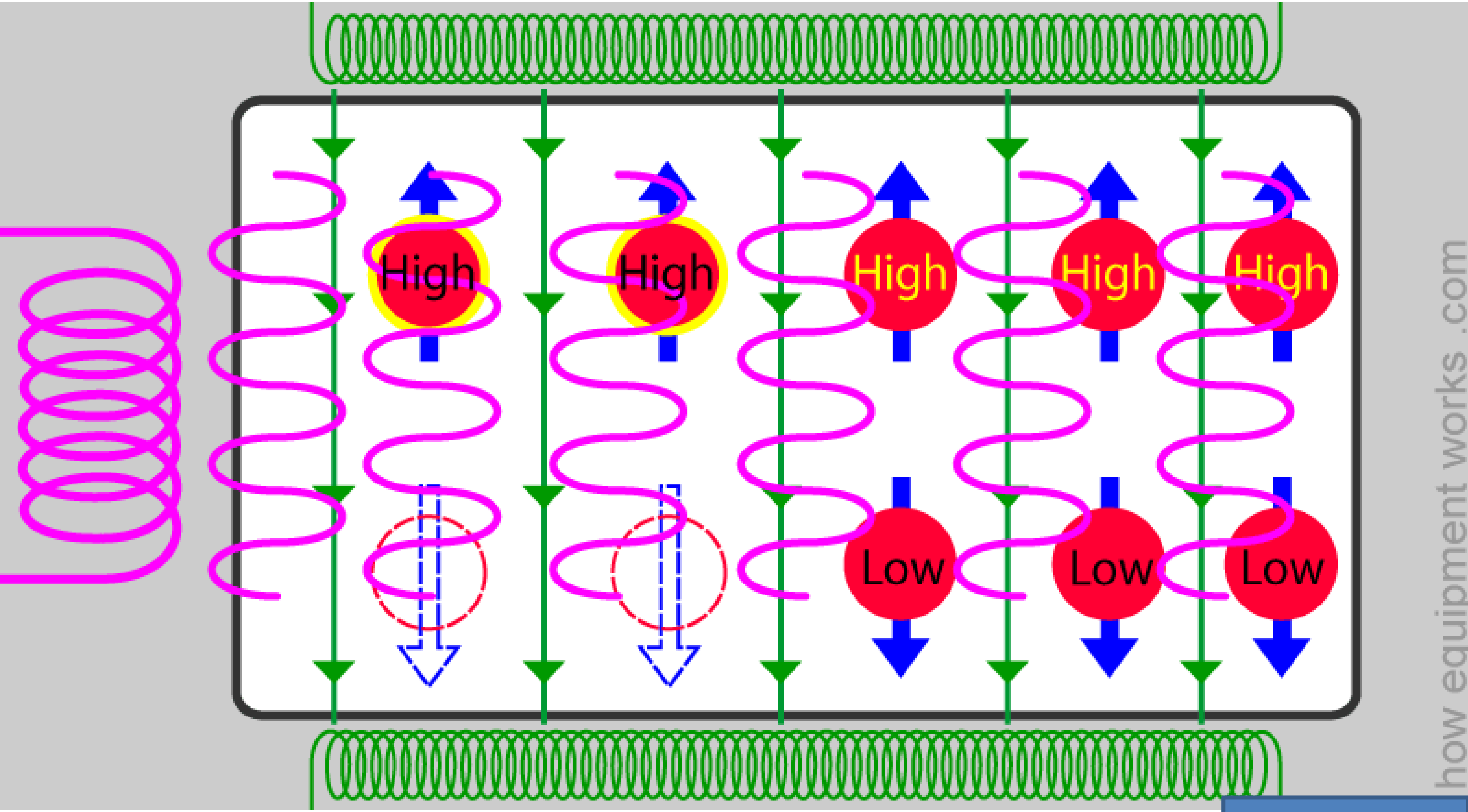
The MRI machine applies a current to this energy producing coil for a short period. During this period, the coil produces energy in the form of a rapidly changing magnetic field (pink waves in diagram below). The frequency (i.e. how often it changes in one second) of this changing field falls within the frequency range commonly used in radio broadcasts. Therefore this energy is often called "radio frequency" energy (RF energy) and the coil is often called an radio frequency coil (RF coil).



Now something fascinating happens to the hydrogen nuclei with low energy. These hydrogen nuclei with low energy absorb the energy sent from the RF coil.

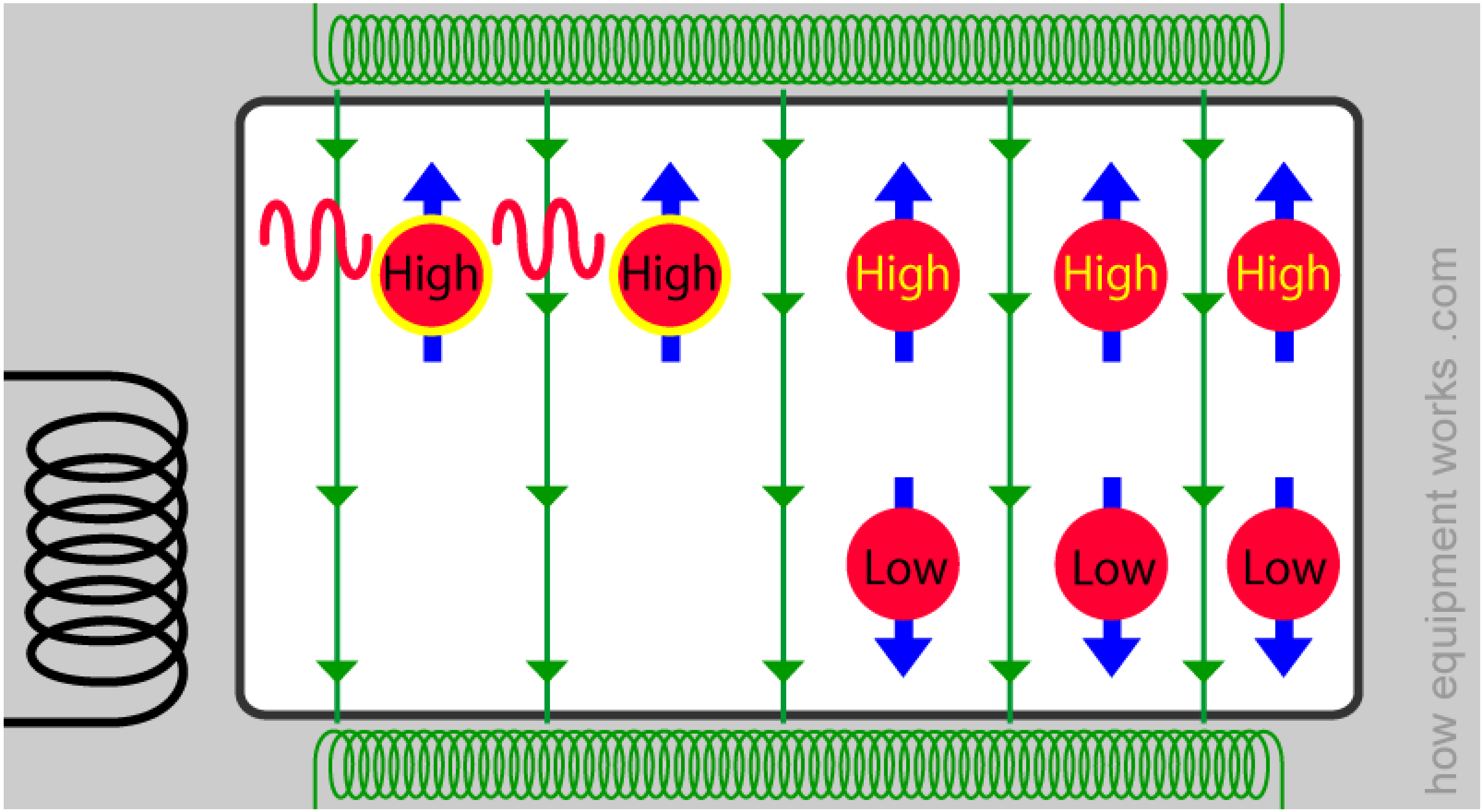


The absorption of RF energy changes the energy state of the low energy hydrogen nuclei. Once the low energy nuclei absorb the energy, they change their spin direction and become high energy nuclei.

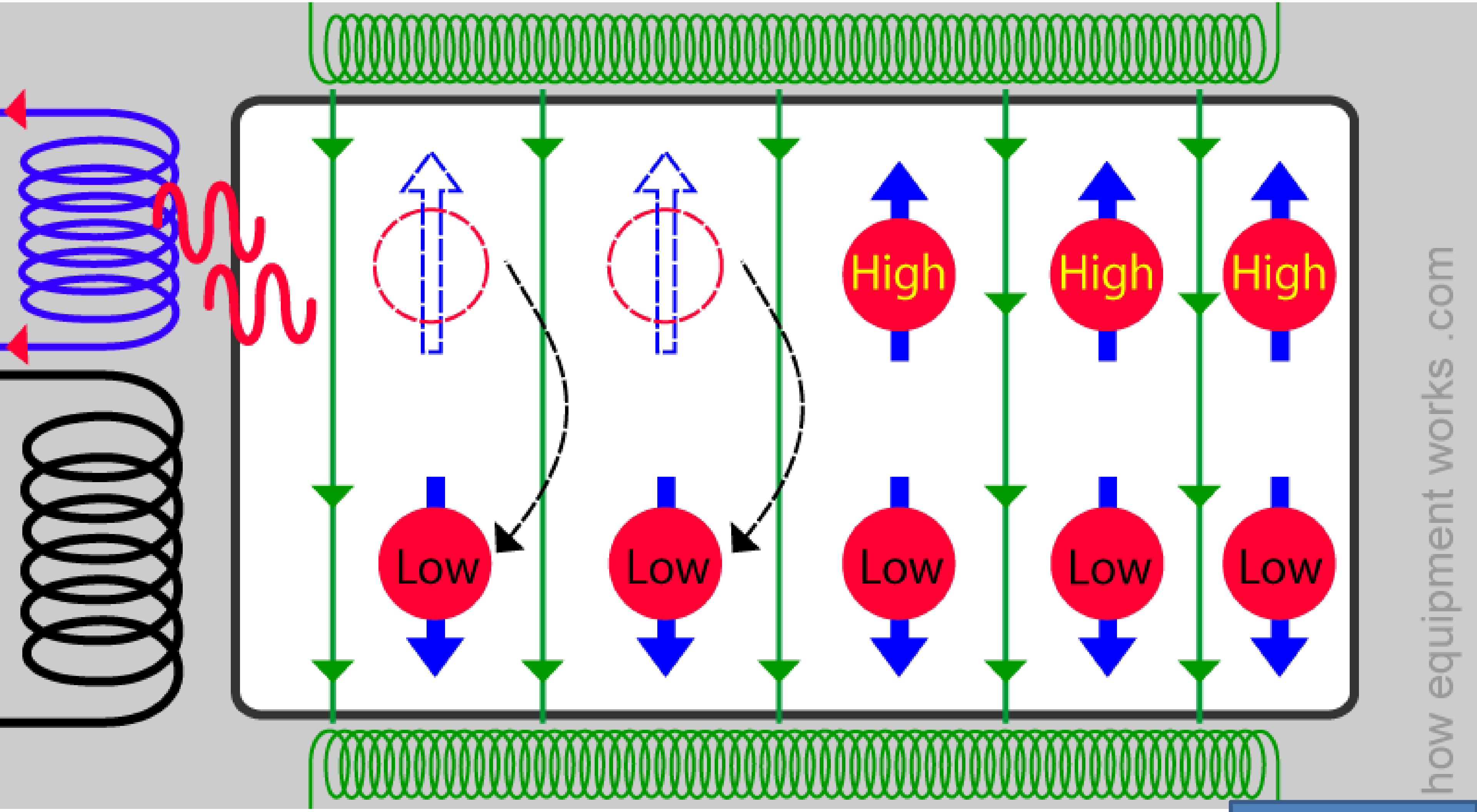


After a short period, the RF energy is stopped

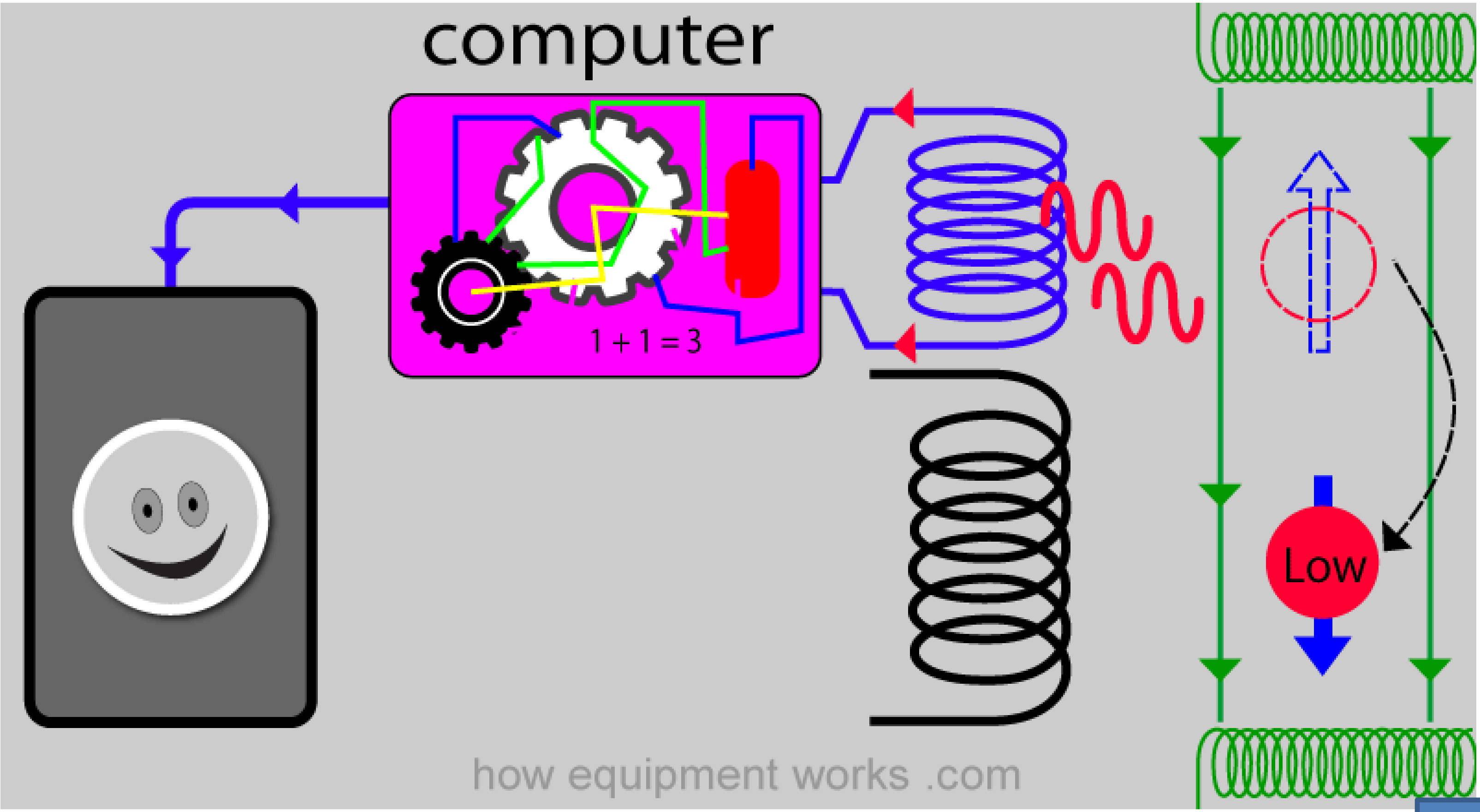
The hydrogen nuclei that recently became ‘high energy’ prefer to go back to their previous, ‘low energy’ state and they start releasing the energy that was given to them (i.e. the previously lazy nuclei want to be lazy again !). They release the energy in the form of waves, which in the diagram below, is shown in red.



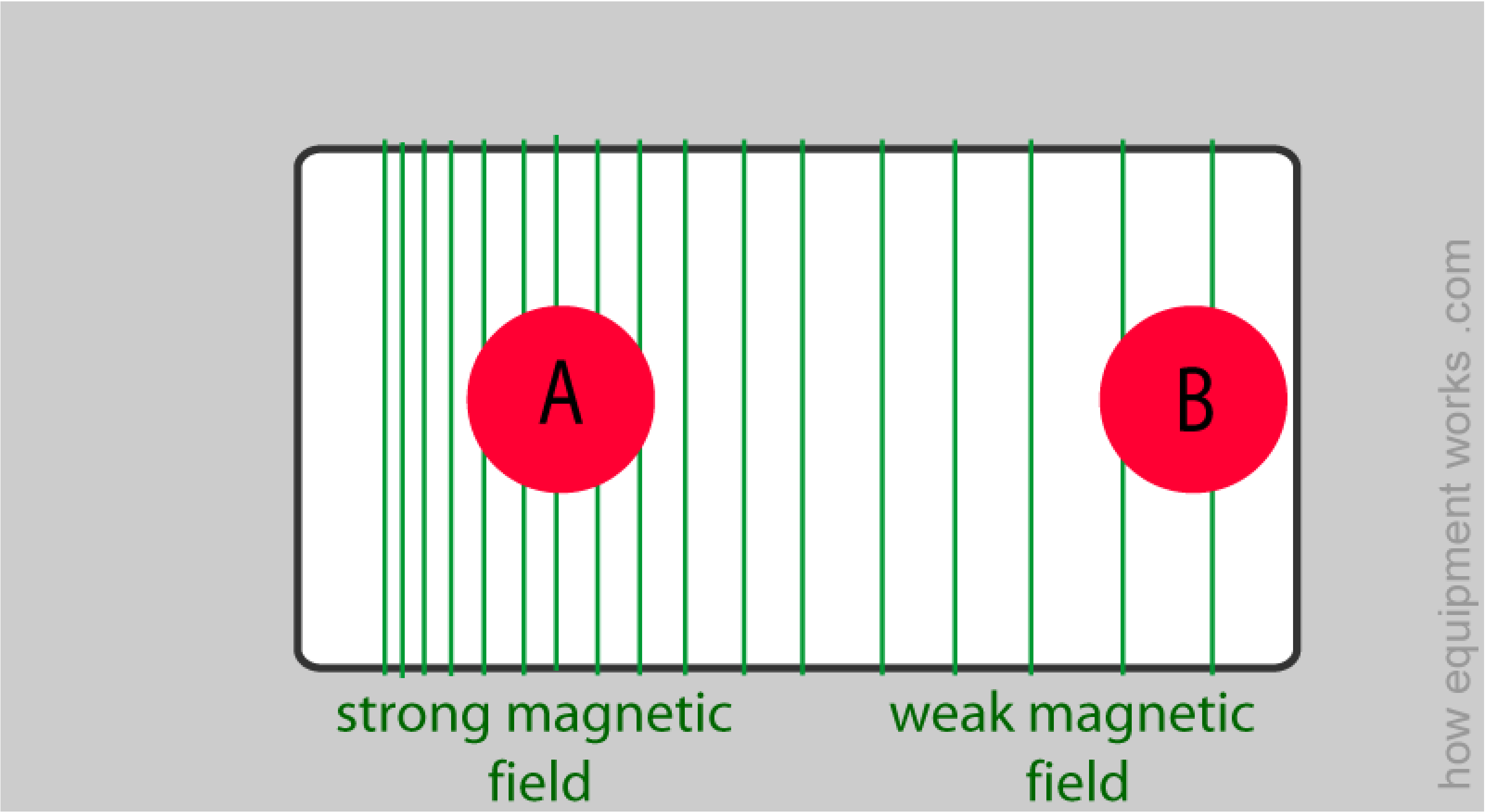
The MRI machine has “receiver coils ” (blue coil shown below) that receive the energy waves sent out by the nuclei. Having given up their energy, the nuclei change their spin direction and return to the low energy state that they were in before.



The computer processes all the information gathered to construct the high quality images. Of course, what have explained to you is a super simplified version of how actually things happen. The real MRI machine works in a much more sophisticated way,.

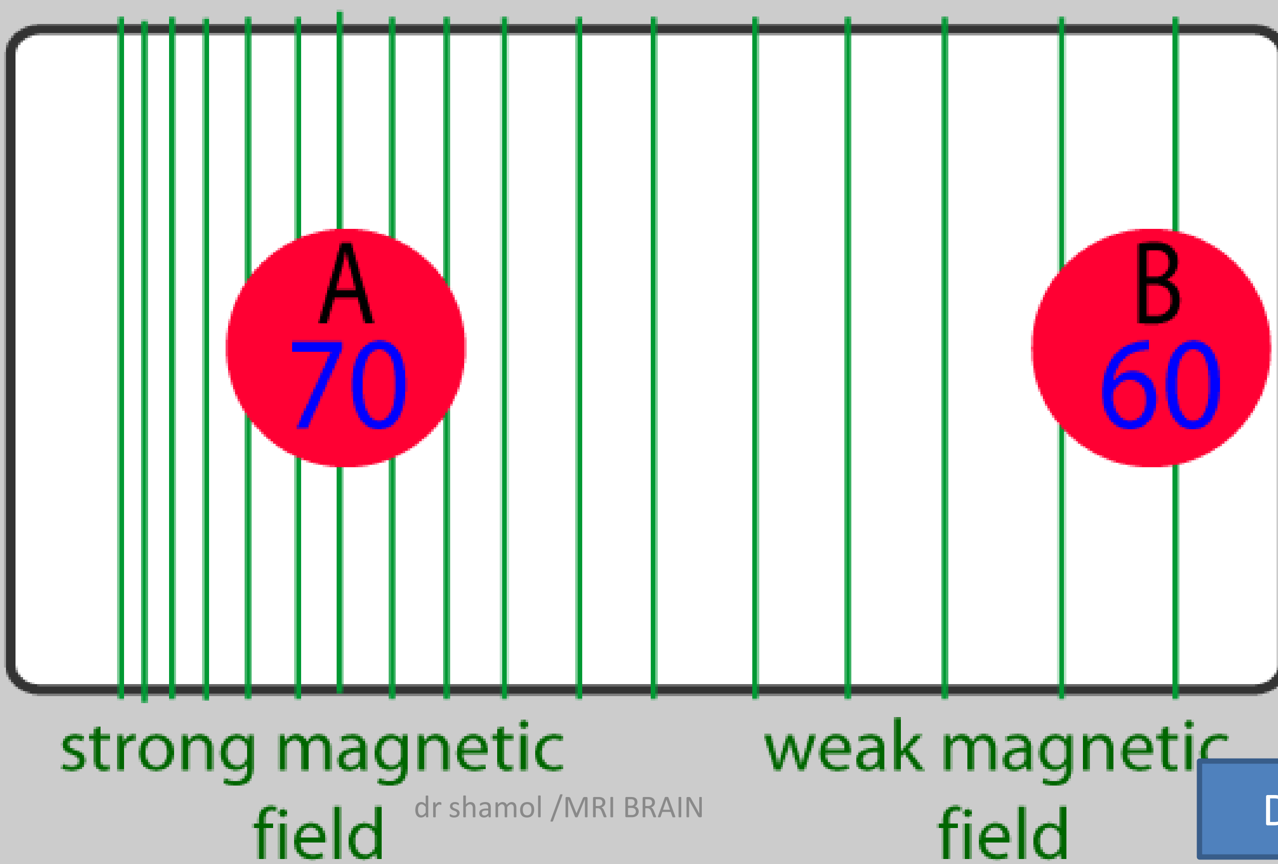


The resonant frequency of an hydrogen nucleus depends on the strength of the magnetic field in the area that it is located in. Higher the strength of the magnetic field, higher is the resonant frequency. Let me explain. Below are two hydrogen nuclei. The hydrogen nucleus ‘A’ is in a stronger magnetic field (represented below with more magnetic field lines) while nucleus ‘B’ is in a weaker magnetic field.

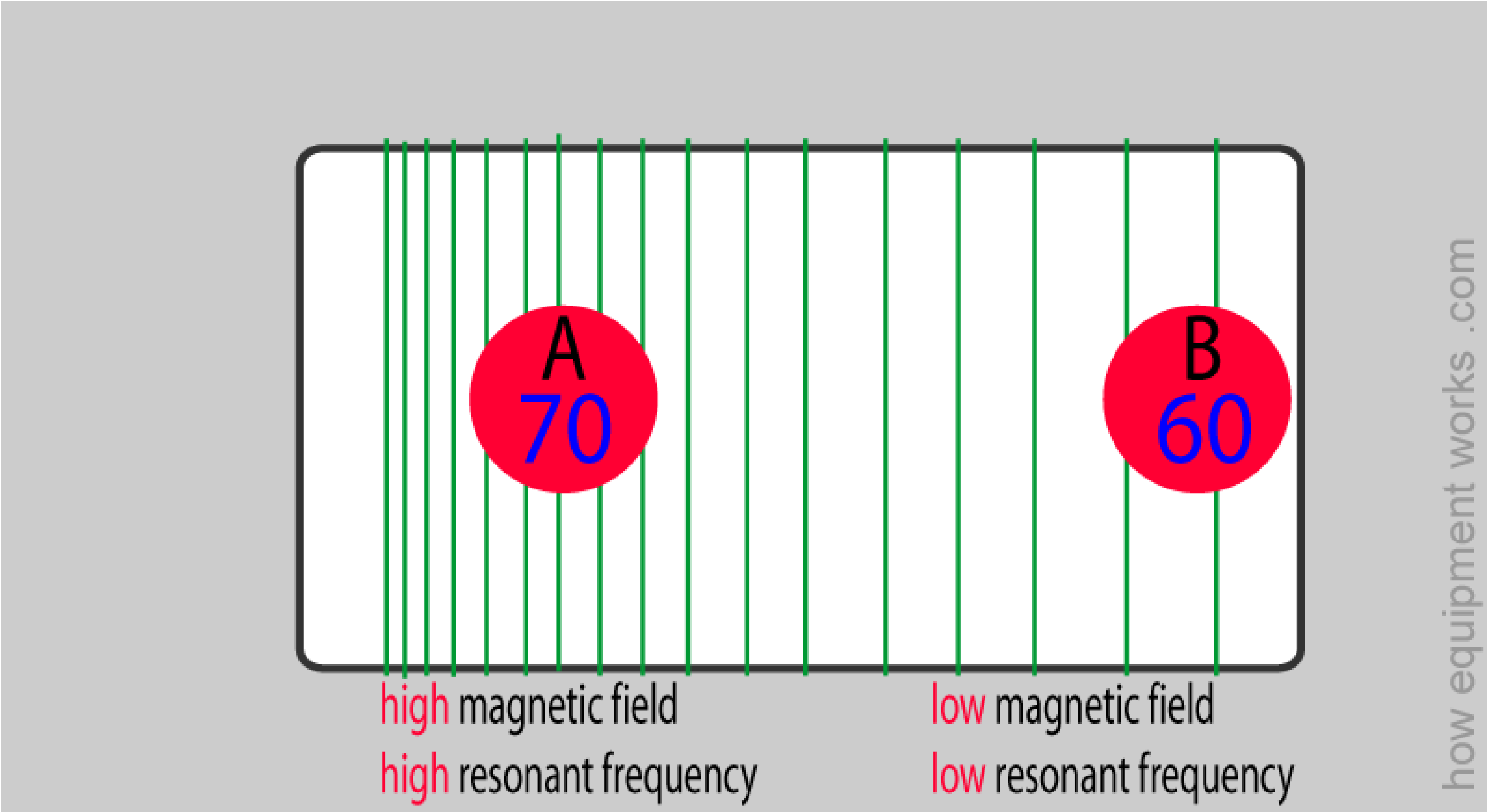


The hydrogen nucleus 'A' is in a stronger magnetic field and therefore has an higher resonant frequency. Just as an example, let us say nucleus 'A' has a resonant frequency of 70 MHz (MHz = mega hertz = millions of cycles per second). I have chosen this number just as an example. The actual values found in MRI scanners will be different.

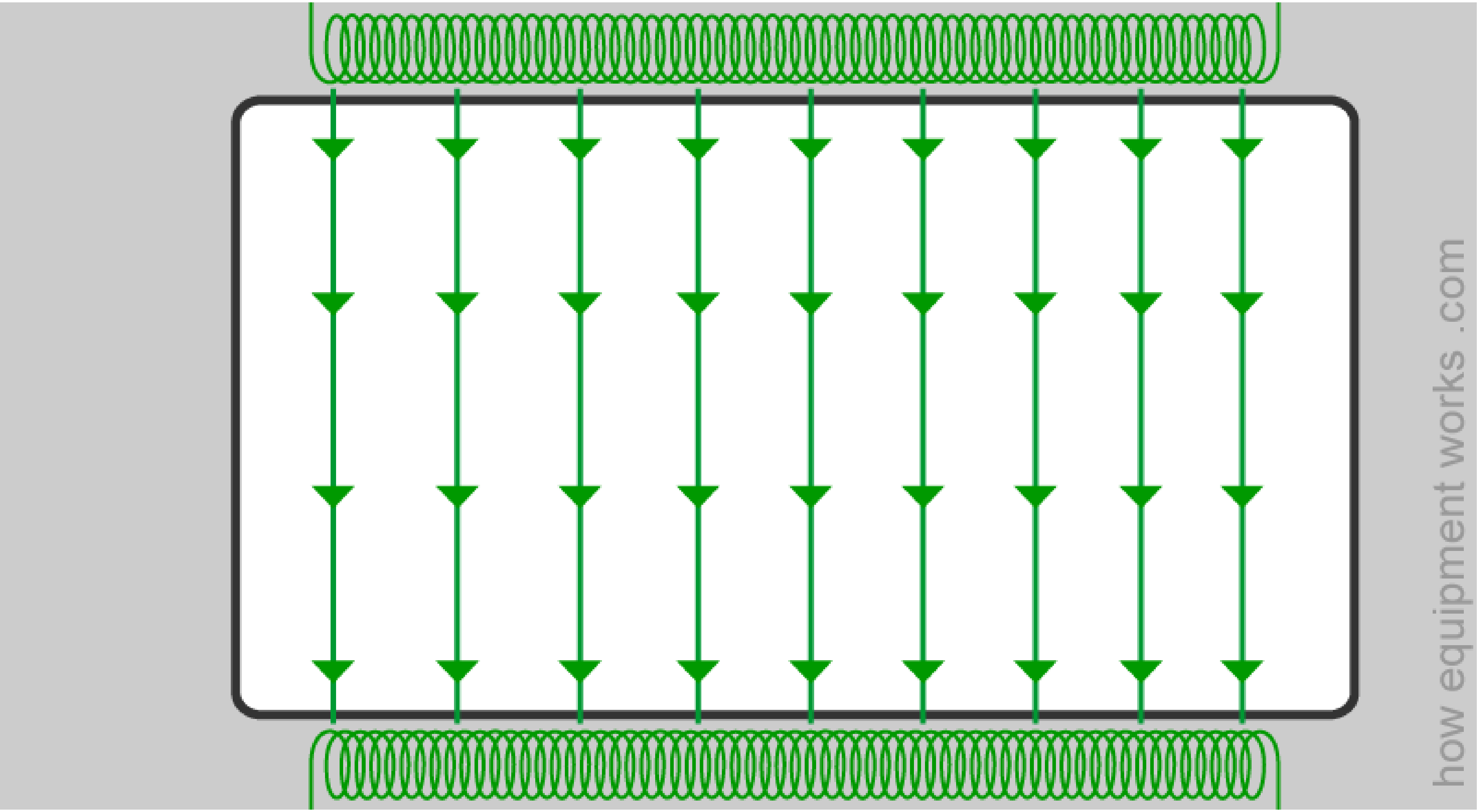
Hydrogen nucleus 'B' is in a weaker magnetic field. Therefore its resonant frequency is lower. For explanation purposes, let us say that it is 60 MHz.



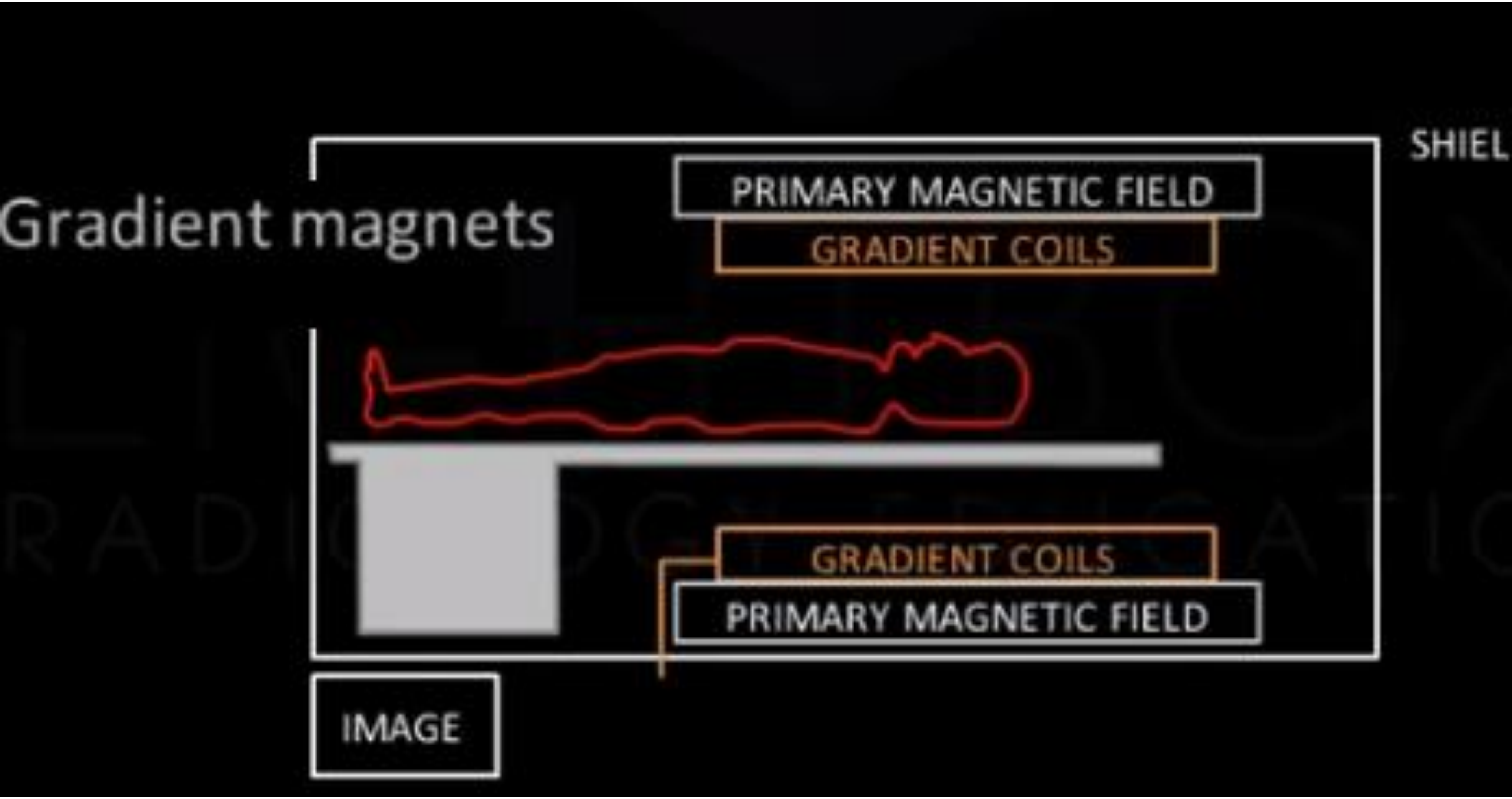
As will be explained to you shortly, this relationship between the magnetic field strength and resonant frequency enables the MRI machine to scan a section of the body at a time.



As mentioned before, there is a strong main magnetic field in the MRI machine. This main magnetic field is uniform throughout. i.e. the strength of the magnetic field is equally strong everywhere.

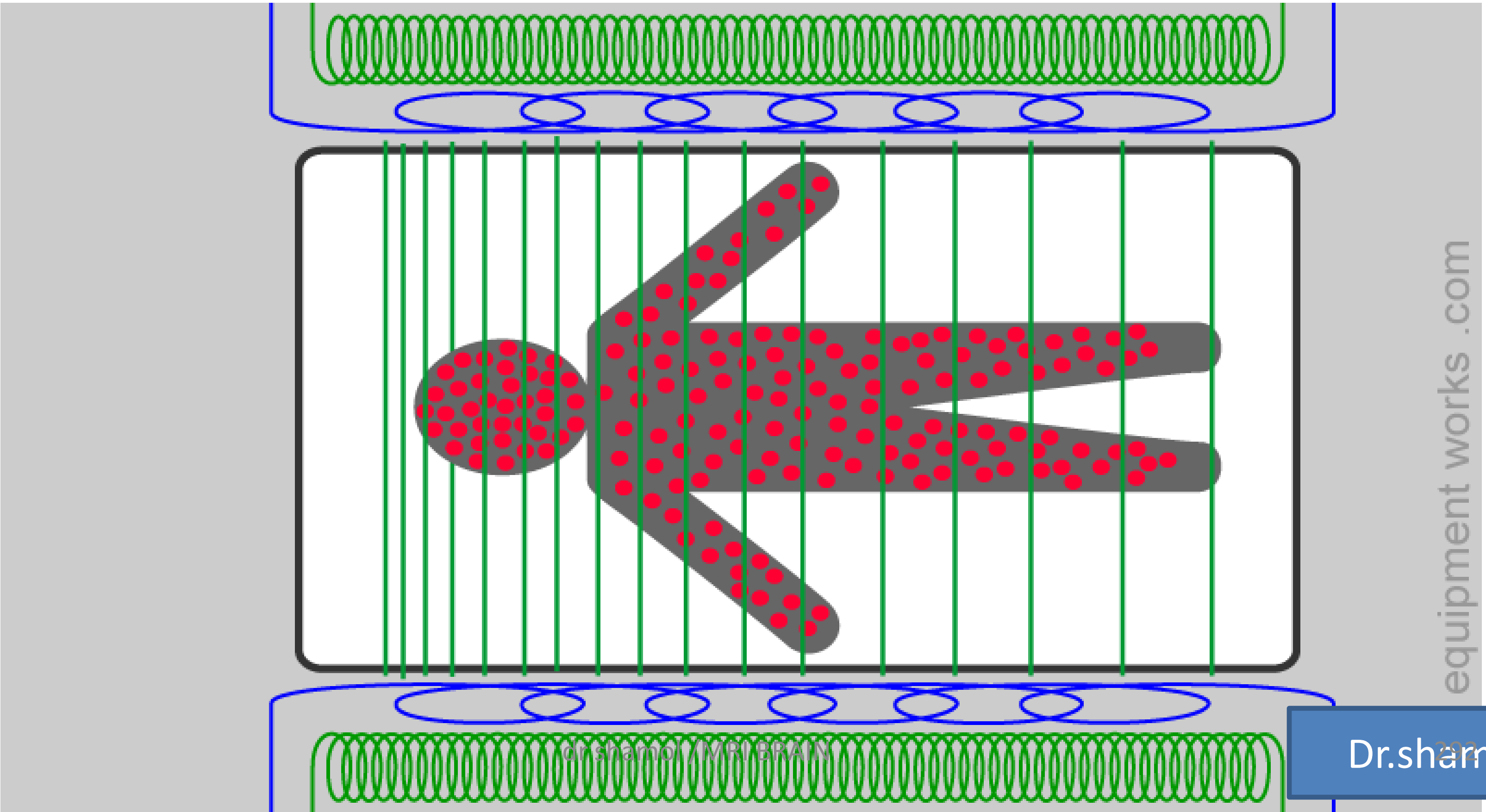


Gradient coil generate secondary magnetic field over primary magnetic field



There are special coils shown in blue in the diagram below that alter the main magnetic field. These coils, ‘modify’ the magnetic field produced by the main magnetic coil (green coil) so as to make one end of the patient have a stronger magnetic field and the other end have a weaker magnetic field.

In the example shown below, as we go from the patients head towards his feet, the main magnetic field becomes weaker. We would now say that there is a ‘magnetic gradient” along the patient. The coils that modify the main magnetic field are called “**gradient coils”.** How these magnetic field gradients are created is fascinating but I will explain that later.



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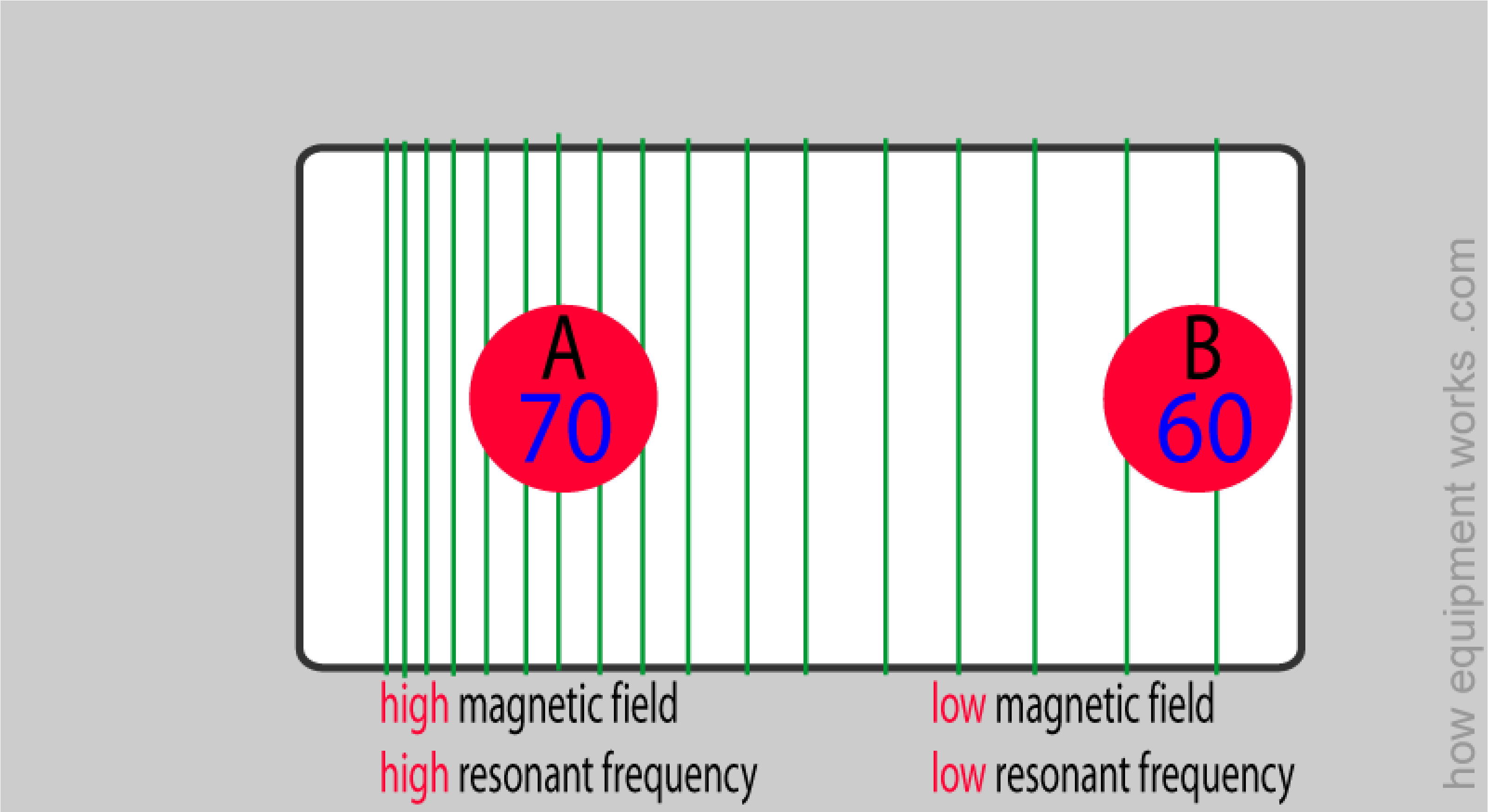
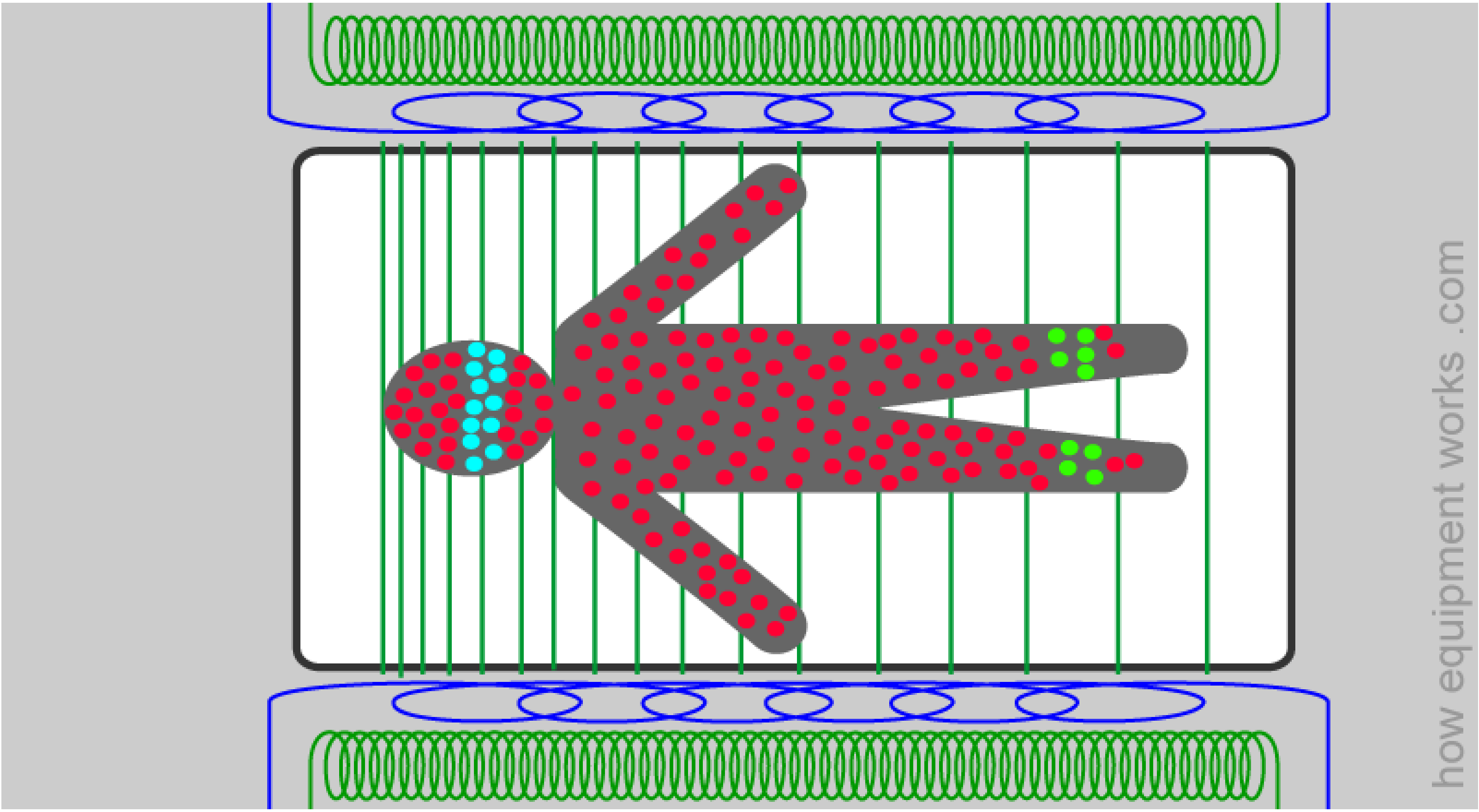
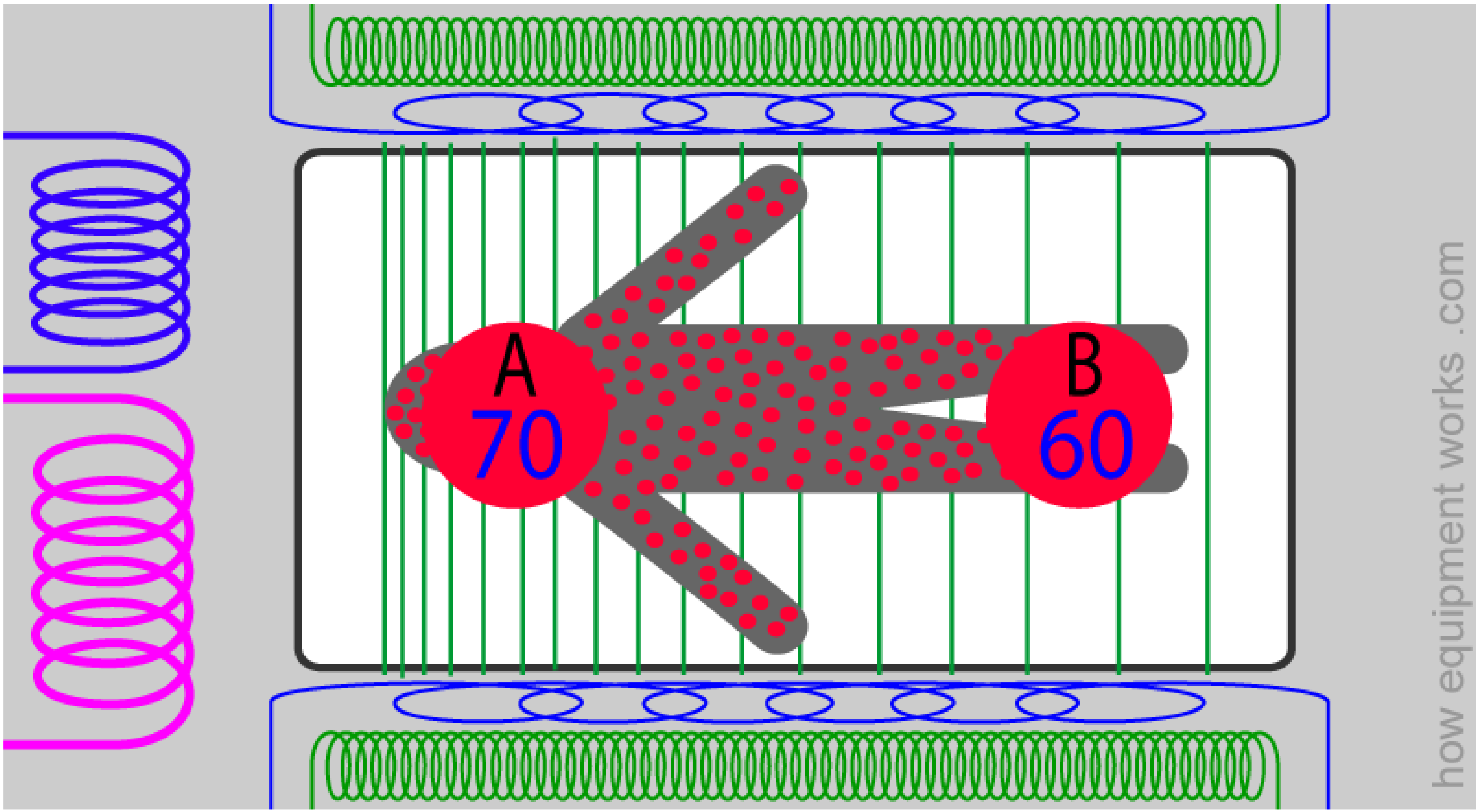
Now you will recall that the resonant frequency of an hydrogen nucleus depends on the strength of the magnetic field in the area that it is located in.



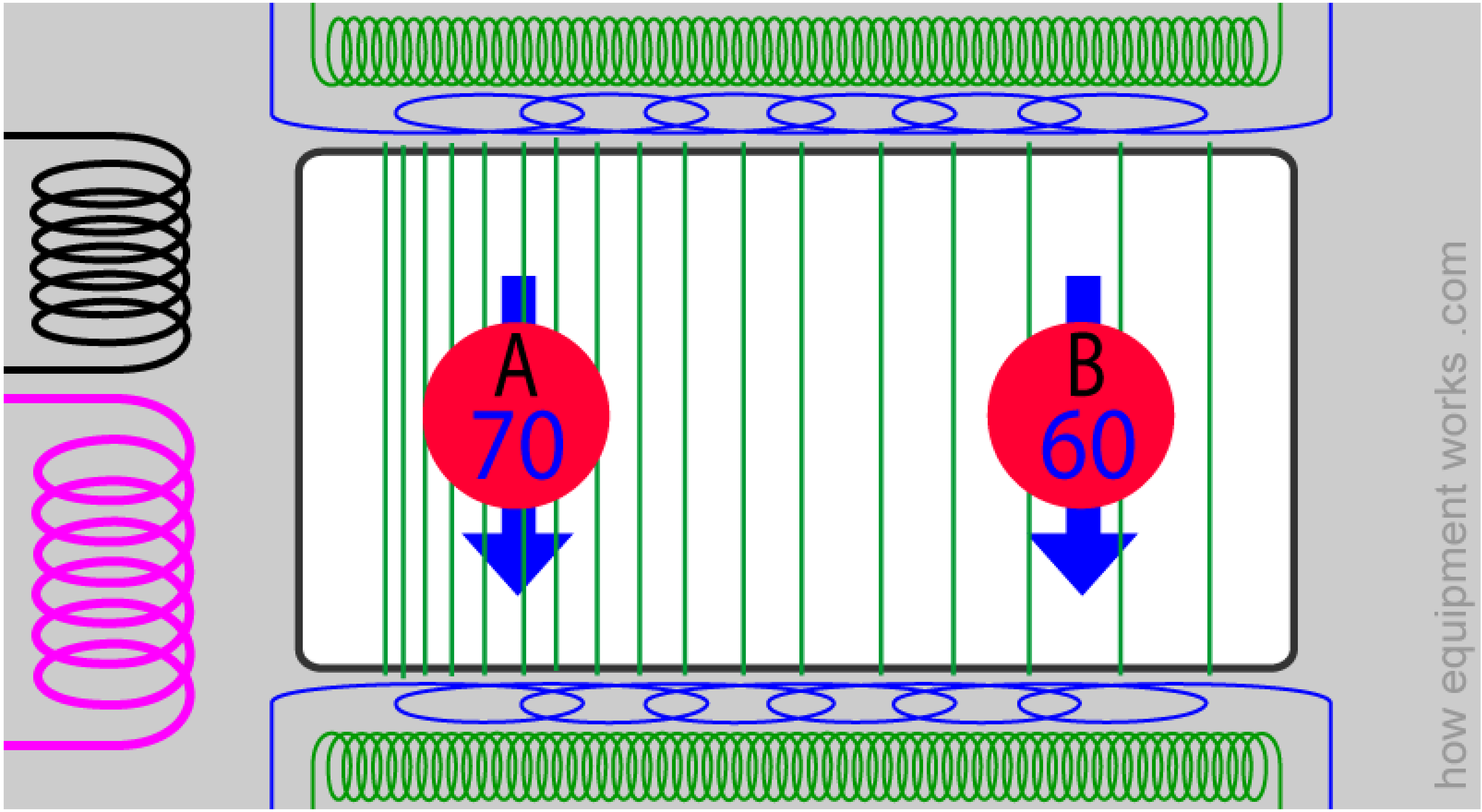
As you have seen before, the gradient coils make the main magnetic field have a gradient : stronger at one end and weaker at the other end. This means that, from the head of the patient to the foot of the patient, the hydrogen nuclei are exposed to different strengths of the magnetic field. At the head of the patient, for an example, the hydrogen nuclei (blue) are exposed to an high magnetic field, and therefore have an high resonant frequency. On the other hand, at the foot of the patient, the hydrogen nuclei (green) are exposed to a low magnetic field and therefore have a lower resonant frequency



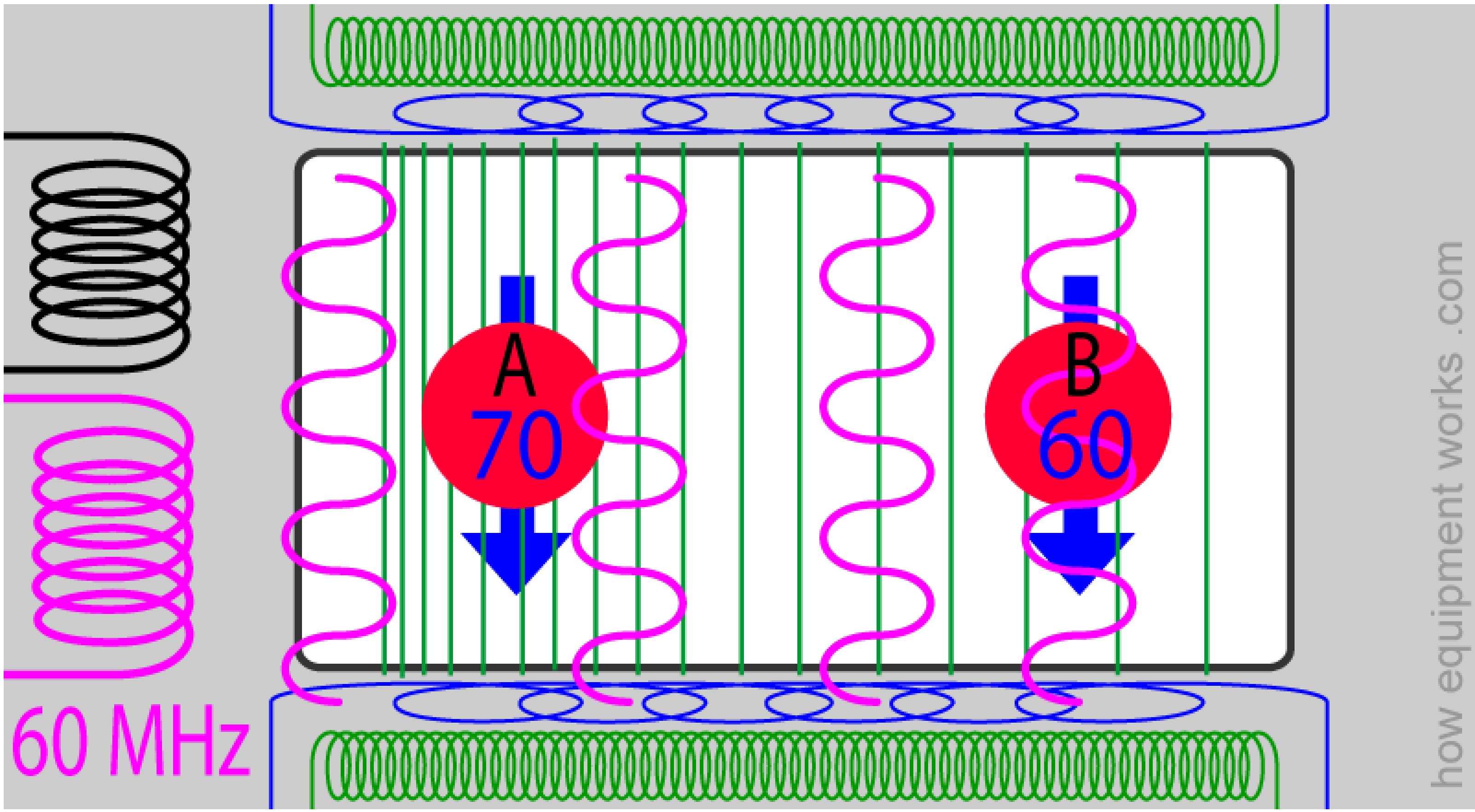
Let us magnify, for clarity, one hydrogen nucleus from the head end (A) and one from the foot end (B). As explained, nucleus A is in a stronger magnetic field and therefore has an high resonant frequency (e.g. 70 MHz). Nucleus B is in a weaker field and therefore has a lower resonant frequency (e.g. 60 MHz).



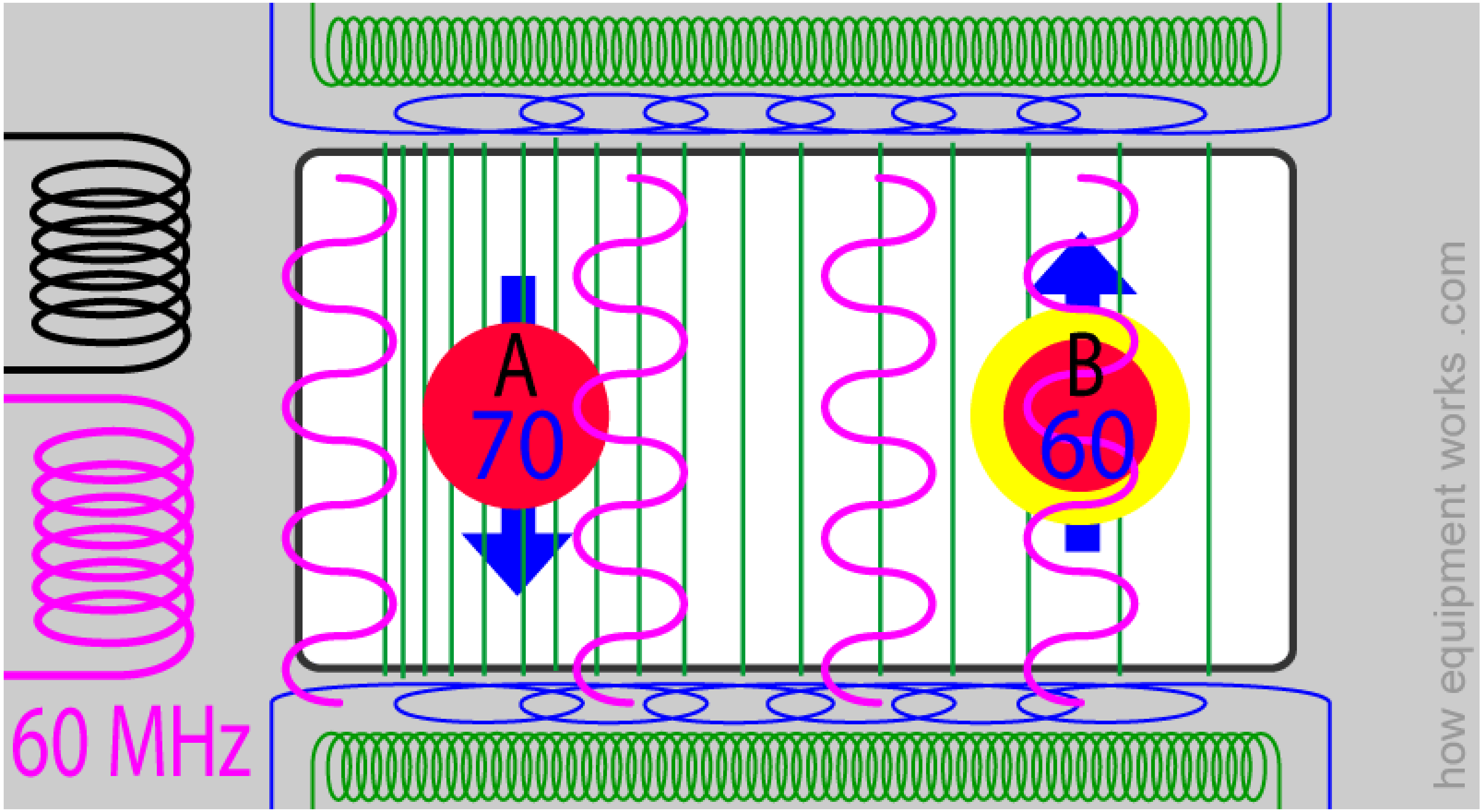
Let us get rid of the patient diagram to make things less cluttered.



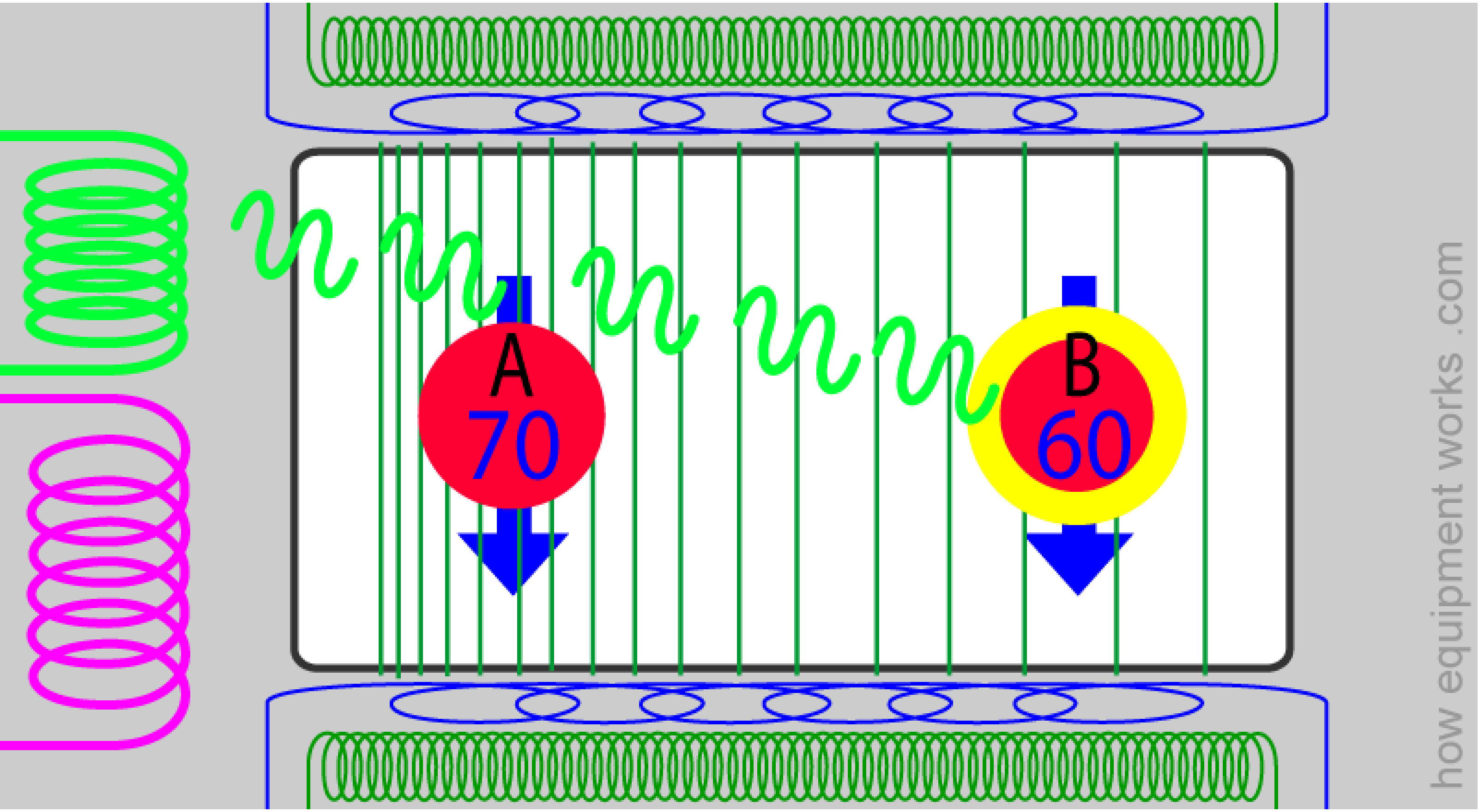
Now you are ready to understand how the MRI machine selects various areas to study. Let us imagine that it wants to image the foot area (i.e. it needs the foot area hydrogen nuclei (B) to respond). It does this by sending RF energy (from the pink RF coil) at the resonant frequency of the nuclei of interest, in this case 60 MHz.



Because the RF energy of frequency 60 MHz is below the resonant frequency of the hydrogen nucleus at the head end of the patient (A), it simply passes this nucleus without getting absorbed. On the other hand, the 60 MHz RF energy exactly matches the resonant frequency of the hydrogen nucleus at the foot of the patient (B). This nucleus therefore absorbs the energy and changes its spin direction to be become an high energy nucleus



Once the RF coil stops transmitting energy, the listening coil (green) listens for a return signal of energy. Since it was only the hydrogen nuclei at the foot end (B) that absorbed energy, it is only these nuclei that now release energy. The other nuclei, e.g. 'A', do not respond, since they did not absorb energy. In this way, the MRI machine is able to get information specifically from the lower limb area of the patient



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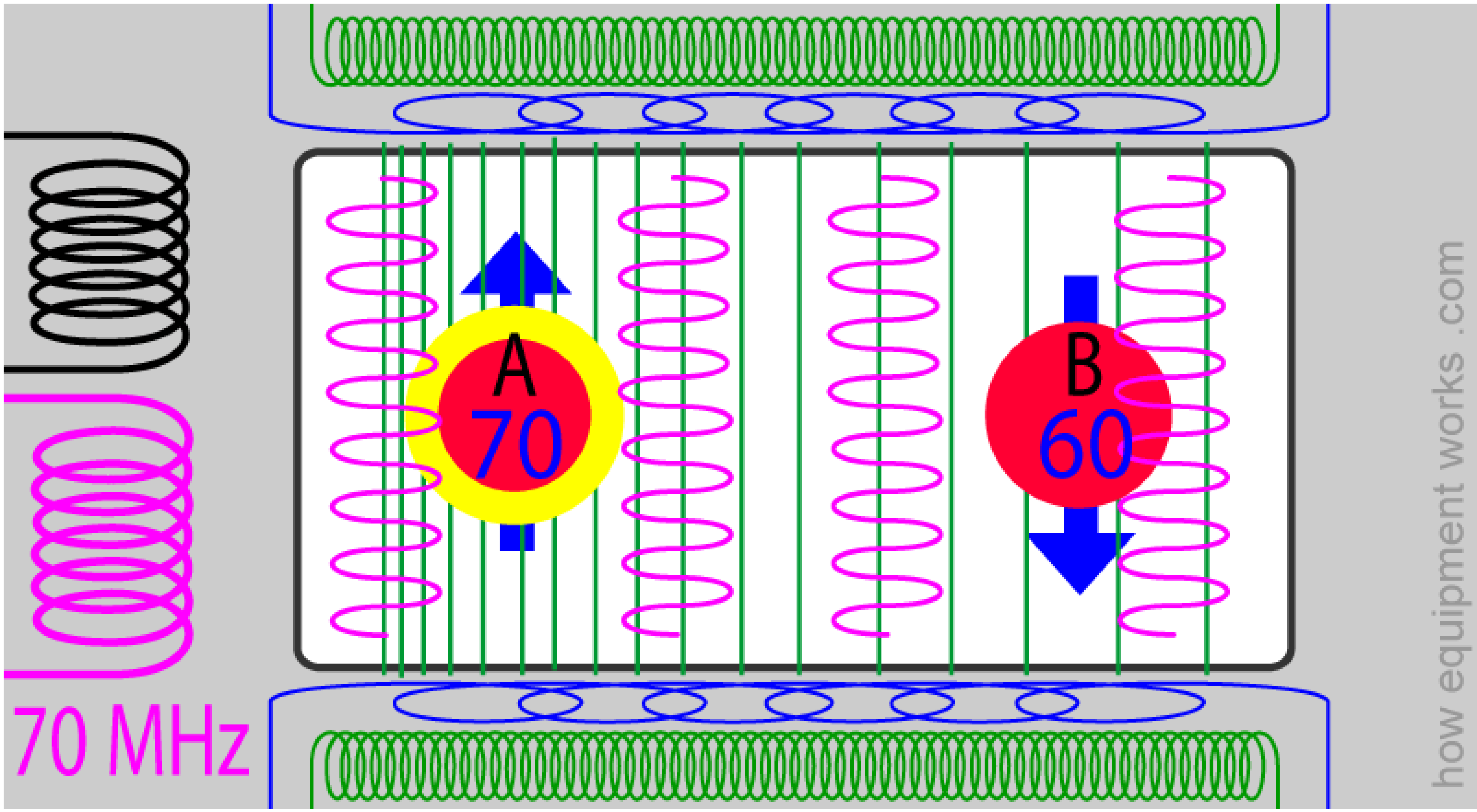
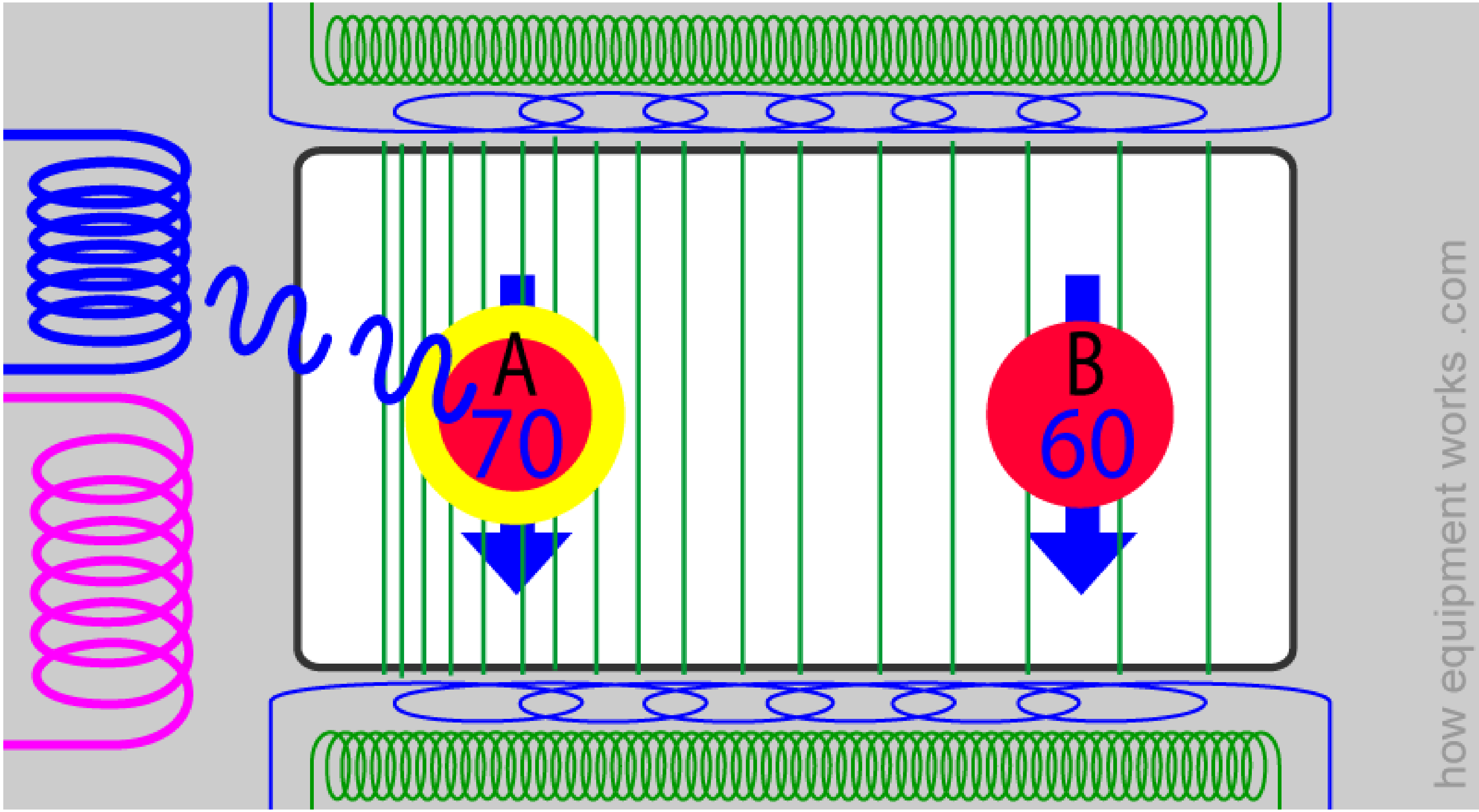
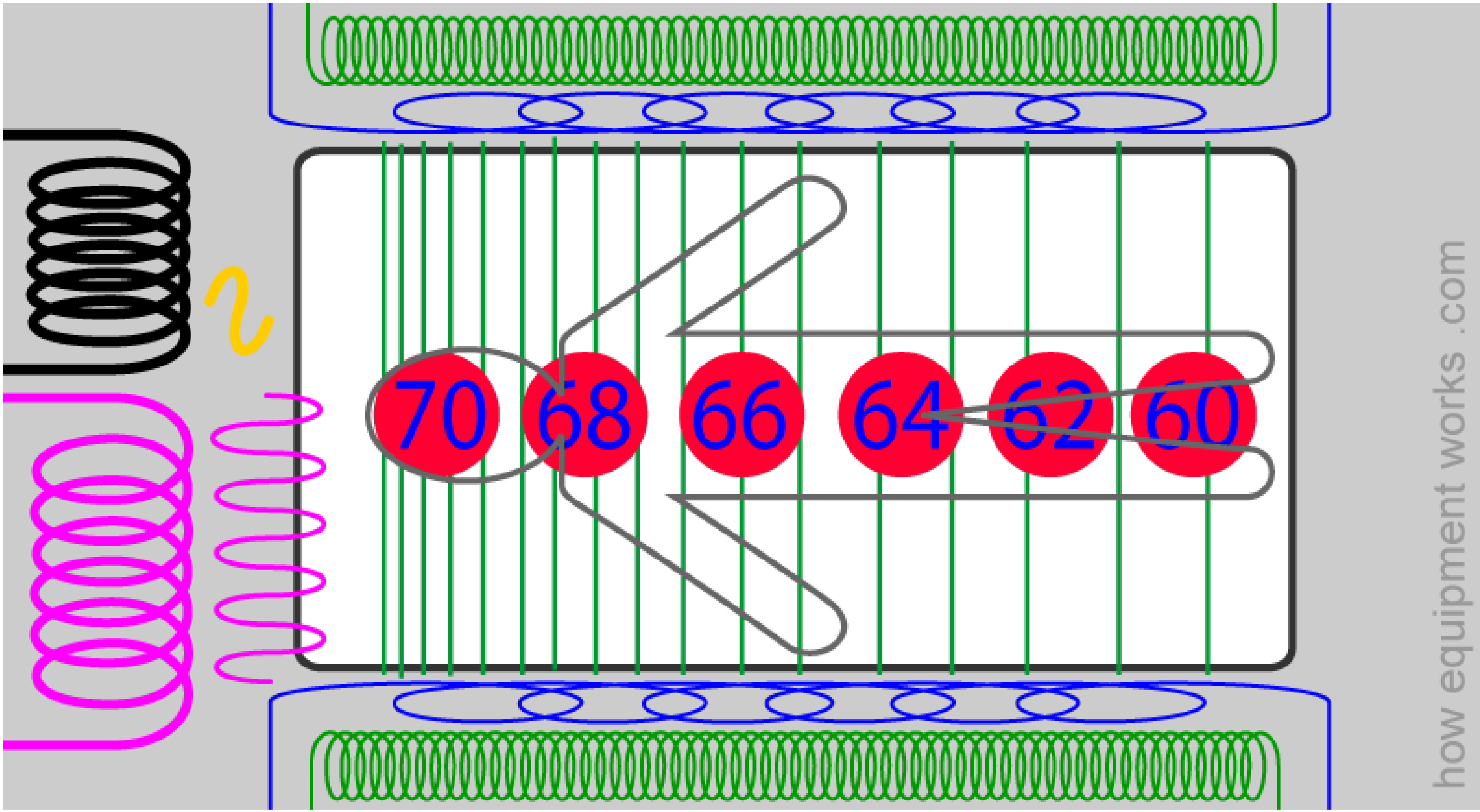
Now let us imagine that the MRI machine wants to image part of the head of the patient i.e. the area where nucleus ‘A’ resides. To do this, the MRI machine changes the emitted RF energy frequency to now match the resonance frequency of nucleus A. Therefore, this time, hydrogen nucleus A absorbs the energy as it matches its resonance frequency, whereas nucleus B at the foot end ignores this energy as it does not match its resonance frequency.



Once the RF coil stops sending energy, this time only nucleus A sends energy back



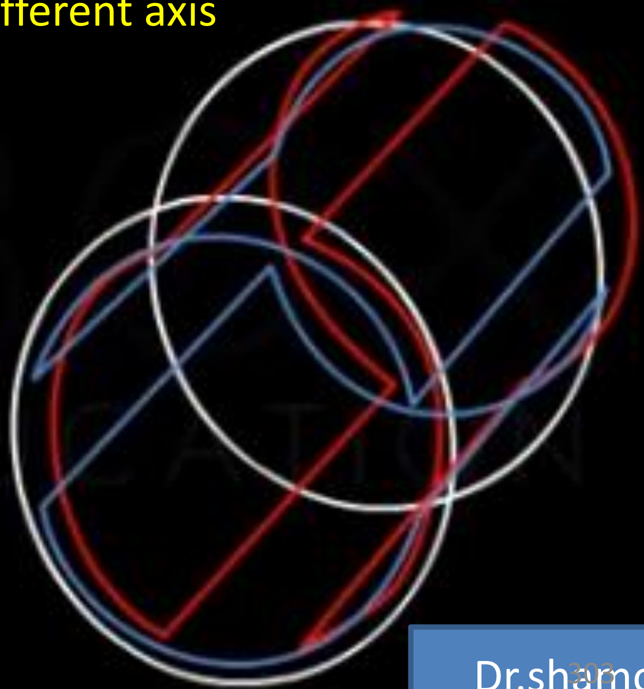
So, by changing the frequency emitted by the RF coil (e.g. 70, 68, 66, and so on), the MRI machine can look for hydrogen nuclei section by section in an orderly manner

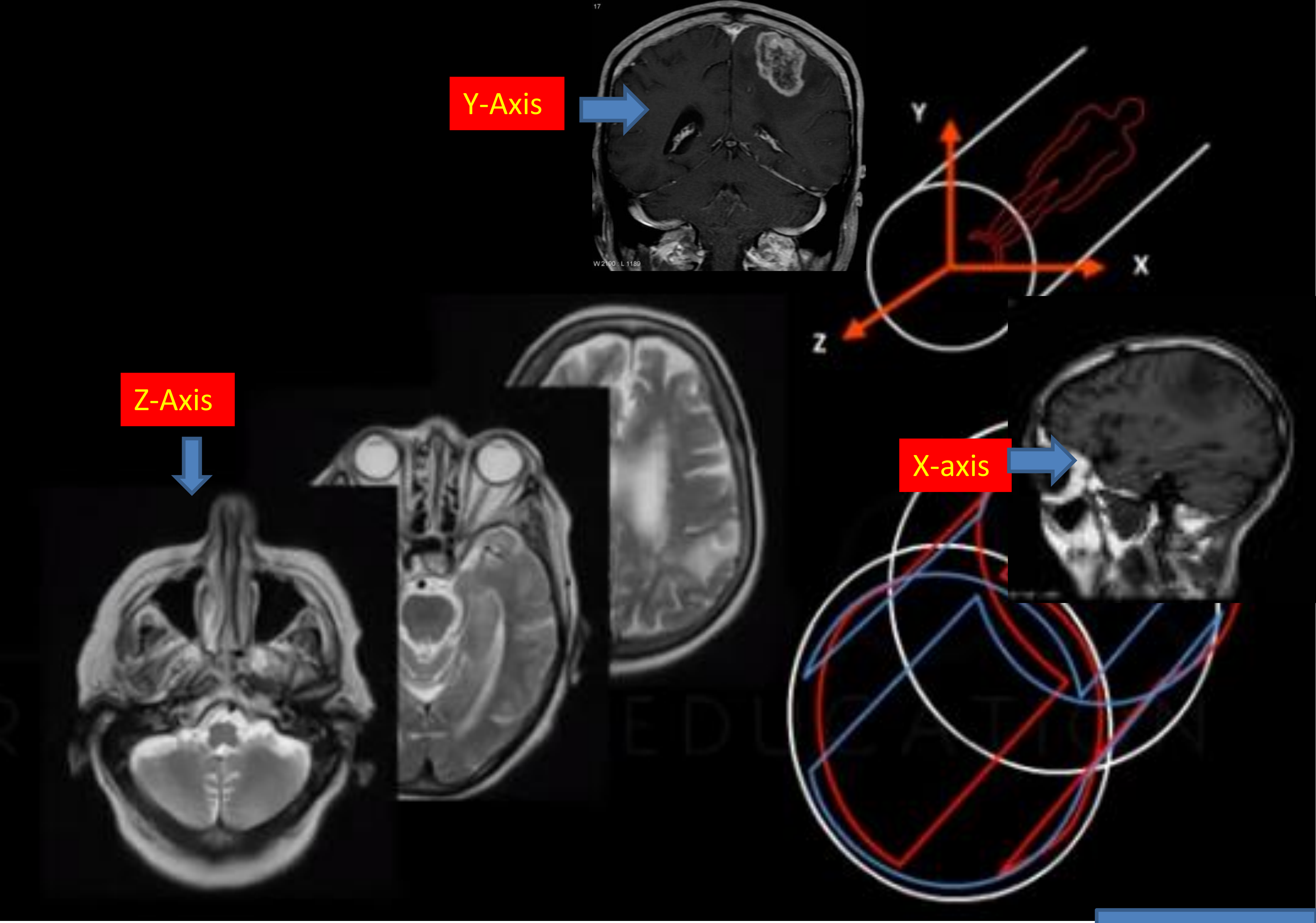


GRADIENT COILS

- Allow spatial encoding for MRI images in the x, y and z axis ie. localisation

Gradient coil are uses for site selection and selection of different axis
Z- axis are for axial image (along the long axis)
Y –for coronal image (along the vertical axis)
X-for sagital image (along the horizontal axis)




Y-Axis
Y
X
Z
Z-Axis
X-axis

RF coil is also used to received signal to produced image

relaxation is the processes by which an excited magnetic state returns to its equilibrium distribution. **By this** process spins release the energy received from a radiofrequency pulse.

Relaxation in longitudinal axis is called longitudinal / T1 relaxation

Relaxation in transverse Axis is called transverse /T2 relaxation